adsorbed on the Ru surface. He proposes that this partial dissociation is a necessary prerequisite for wetting, at least on Ru(0001).

If this is correct, then theory and experiment are reconciled. H or D scatter electrons very weakly and contribute very little to the LEED determination, which mainly yields the locations of the O atoms. And for these, the Feibelman structure agrees with the LEED data. But other findings are difficult to reconcile with partial dissociation. For example, thermal desorption spectra do not show any evidence of two species and of recombination (*6*). The calculations also do not explain why water should partially dissociate below 150 K but fully associate back to the desorbing molecule above 170 K.

A similar layer on Pt(111) (*7*), which vibrational spectroscopy suggests to consist of intact water molecules, has LEED curves identical to that of a layer supposed to consist of OH (*8*). Hence, either the "intact" layer is mixed, or the structures of intact and partially dissociated layers are indistinguishable. Mixed H_2O + OH layers certainly can exist (*9*); the question is whether they are the only stable layers.

A definitive experiment to resolve this question is difficult to devise because the only difference between the structures in panels A and B is that in panel B, one H atom has been moved from being bound to O to being bound to Ru. Neutron scattering cannot be used to study surface species, and other structural tools do not respond sufficiently to H; most spectroscopic techniques cannot distinguish between adsorbed H_2O and OH. On the theory side, all is not so well either. Even the best available DFT procedures cannot model van der Waals interactions reliably; their absence might have tipped the energy balance in the present case. Also, the adequacy of DFT to model hydrogen bonds might be debatable because the calculations do not treat the atoms quantum mechanically (the hydrogen atoms are given infinite mass; their zero point energy is taken care of by rescaling the energy scale).

Two focal points of surface science—the comparison of theory and experiment, and the understanding of an extremely interesting particular system—coincide to make water on metal surfaces an interesting and challenging issue. Feibelman's study points toward an interesting possible solution and will trigger further experimental and theoretical work. Clearing up the issue of how the first water layer binds to a transition surface will require—and lead to—improvements in both theory and experiment of surface interactions.

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PERSPECTIVES: PLANT BIOLOGY

Prime Time for Cellulose

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Every year, plants make more than 10^{11} metric tons of cellulose, the chain of glucose residues that is the principal component of the plant cell wall. However, biochemical dissection of the three steps of cellulose synthesis—initiation of the sugar chain, elongation, and termination—is still in its infancy. A notable advance has been the recent identification of the *CesA* genes that encode protein subunits of cellulose synthase, the rosette-shaped enzyme complex in the plant cell plasma membrane that synthesizes cellulose (*1–3*). Despite this advance, it has still proved difficult to coax plant extracts to make cellulose in the test tube—instead they make callose, a wound-response polysaccharide. It is possible that cellulose synthase is particularly fragile, or requires an intact membrane environment for activity. A big leap forward comes on page 147 of this issue with the identification by Peng *et al.* (*4*) of the lipid sitosterol-β-glucoside (SG) as the primer for cellulose synthesis in plants.

The structural backbone of plant cell walls comprises crystalline microfibrils of cellulose, each composed of 36 β-(1,4)–linked glucan chains. Noncrystalline (soluble) cellulose accumulates in response to the herbicide CGA 325′615 or plant mutations that block cellulose synthesis or activity of the Korrigan cellulase enzyme (*5*, *6*). In the new work, Peng *et al.* show that digesting noncrystalline cellulose with cellulase releases not only CesA proteins but also small amounts of a sitosterol lipid linked to glucose (*4*, *5*). Further metabolic studies led the authors to propose a biosynthetic pathway for cellulose that starts with transfer of a glucose residue from the soluble cytoplasmic substrate uridine 5′-diphosphate (UDP)–glucose onto sitosterol to form SG on the inner face of the plasma membrane (see the figure). SG then acts as a primer, initiating the polymerization of glucan chains catalyzed by CesA proteins of the cellulose synthase complex. The result is formation of lipid-linked oligosaccharides called sitosterol cellodextrins (SCDs). Formation of SG and SCDs is blocked by the herbicide DCB (2,6-dichlorobenzonitrile), which targets cellulose synthesis. The cellodextrins may be cleaved from the sitosterol primer by Korrigan cellulase, the active site of which is predicted to be located on the outer face of the plasma membrane. This model defines the point at which the primer is transferred through the energy barrier of the membrane lipid bilayer, although how this happens is unknown. Further elongation of the cellodextrins, catalyzed by the same or different CesA proteins, produces the glucan chains of cellulose, which then coalesce into microfibrils.

Peng *et al.* (*4*) replicated part of the plant cellulose synthesis pathway in transgenic yeast, the perfect model organism because they do not make cellulose. Yeast containing a single *CesA* gene, in this case *GhCesA-1* from cotton, make SCDs, whereas control yeast do not. The investigators thus were able to identify SCDs as lipid intermediates in the initiation of cellulose synthesis. The transgenic yeast, however, do not make cellulose, so some other part of the synthetic machinery is clearly missing. It is still a mystery how cellulose chains containing several thousand β-(1,4)–linked glucose residues are extruded into the walls of plant cells and then are terminated.

Plants cannot survive without cellulose, which may account for why they possess an extended family of *CesA* genes. Members of this family are expressed at different times and do different things; some pairs of *CesA* genes are even switched on together (*7–9*). Thus, plants with mutations in a particular *CesA* gene show reduced cellulose synthesis only in certain cell types or at specific life-cycle stages, resulting in distinct phenotypes such as root swelling or an irregular xylem.

The new work raises the intriguing pos-

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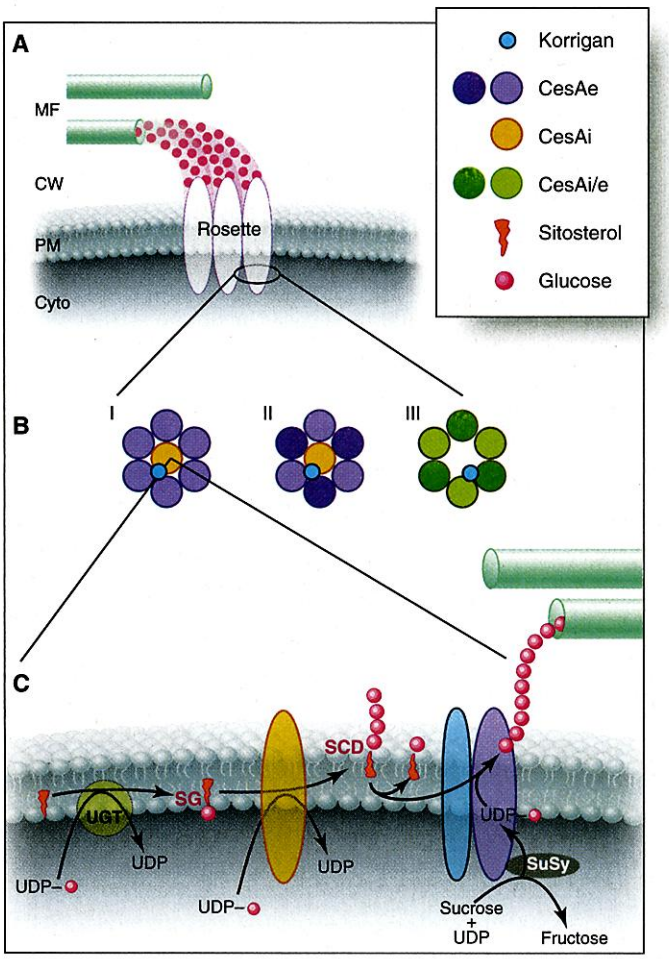
sibility that plants contain many *CesA* genes so that one CesA protein could catalyze chain initiation on the SG primer, whereas another could catalyze elongation (see the figure). Alternatively, different CesA proteins might be required for assembly of the rosette complex, or for the elongation reaction itself (*9*). Intuitively, one would presume that, compared to the CesA protein responsible for chain elongation, much less would be required of the CesA protein that adds glucose units to the SG primer, or of Korrigan cellulase that cleaves cellodextrins from the primer. Perhaps the six "elementary particles" that comprise the cellulose synthase rosette each contain just one molecule of the initiating CesA and Korrigan cellulase (see the figure). Functional genomics should help to unravel the arrangement of proteins in the rosette.



A plant primer. The cellulose synthase rosette in the plant cell plasma membrane. (**A**) Longitudinal view of the rosette composed of six elementary particles during elongation of cellulose microfibrils (MF). Sucrose synthase (SuSy) on the cytoplasmic face of the plasma membrane (PM) may channel UDP-glucose to the 36 growing glucan chains, which are extruded into the plant cell wall where they coalesce to form microfibrils. (**B**) Possible substructures within one elementary particle of the rosette. (i) The particle contains six elongating polypeptides (CesAe), one initiating polypeptide (CesAi), and one copy of Korrigan cellulase. (ii) The particle contains three copies each of two types of elongating polypeptide (CesAe), one initiating polypeptide (CesAi), and one copy of Korrigan cellulase. (iii) The particle contains three copies each of two types of polypeptide that both initiate synthesis and promote chain elongation (CesAi/e), and one copy of Korrigan cellulase. (**C**) Initiation of cellulose synthesis. UDP-glucosyl transferase (UGT) transfers a glucose residue onto a sitosterol molecule on the cytoplasmic face of the plasma membrane, forming sitosterol-β-glucoside (SG). The short glucose chain is extended with UDP-glucose by an initiating CesAi subunit to form an SCD, which "flips" to the outer face of the plasma membrane. The cellodextrin chain is then cleaved by Korrigan cellulase, binds to the elongating CesAe, and is extended into a glucan chain by addition of UDP-glucose provided by sucrose synthase. [Adapted principally from (*9*) and (*16*)]

Peng and colleagues (*4*) transformed yeast with *GhCesA1*, a gene involved in cellulose synthesis in cotton, and showed that yeast could produce SCDs in their plasma membrane upon addition of SG and UDP-glucose. The next step will be to engineer yeast to express other *CesA* genes, either alone or in combination, and to distinguish which are involved in chain initiation and which in chain elongation. The trick will be to see whether yeast can be coaxed to synthesize either crystalline or noncrystalline cellulose. Of course, yeast that make cellulose will not lead to paper (or blue jeans) being produced in fermenters, but the development of reconstituted biochemical systems for synthesis of cell wall polysaccharides will be a huge leap toward understanding how a functional cell wall is assembled.

Diverse organisms use lipid primers for cellulose synthesis. For example, in *Agrobacterium*, cellulose synthesis may involve transfer of lipid-linked cellodextrins to a growing glucan chain after cleavage by a cellulase (*10*). Some eukaryotes, such as the slime mold *Dictyostelium*, have only one *CesA* gene yet make cellulose; that this organism has enzymes that glucosylate sterols suggests that it may also use SG as a primer (*11*, *12*). Plant pollen tubes make cellulose using CslD, a cellulose synthase–like protein related to CesA. As pollen tube growth is sensitive to the herbicide DCB, CslD may also use SG as a primer (*13*).

How are other β-glucans made? One hypothesis is that cellulose synthase defaults to making the β-(1,3)–glucan callose when its rosette structure is destroyed during disruption of the plasma membrane (*1*). This remains plausible despite recent evidence that developmentally regulated callose synthases, such as that catalyzing callose synthesis in pollen tubes, are encoded by a different set of genes (*13*). Sitosterol does not appear to be needed for wound-activated synthesis of callose at the plasma membrane. Synthesis of a mixed-linkage β-(1,3;1,4)–glucan in grasses and related plants may also involve a CesA protein, or one of the closely related CesF proteins newly defined in rice (*3*, *14*, *15*).

Mapping the active sites of cellulose synthase and of other synthases should clarify whether each type of glucan linkage is catalyzed by a different enzyme or by multiple active sites on the same enzyme. In addition, such experiments should reveal whether chain initiation and elongation require different CesA proteins. The identification by Peng and co-workers of a primer for cellulose synthesis in plant cell walls is a big step along this pathway of discovery.

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17. We thank E. Newbigin and C. Vergara for helpful comments.