

synchronous with interstadials (multimillennial warm periods within a broadly glacial period) in the Northern Hemisphere, but lagged sea surface temperatures by several thousand years during glacial-interglacial transitions. In contrast, sea surface temperatures changed at times when solar insolation received in tropical latitudes changed drastically as a result of changes in Earth's orbit.

These findings demonstrate the importance of considering both atmospheric and oceanic circulation systems that can force changes at different time scales in disparate locations. Consider, for example, the Younger Dryas (12,700 to 11,500 years ago), when Earth's climate system reverted back to near-glacial conditions during the period of post-glacial warming. During this time, Weldeab *et al.* observe a substantial steplike reduction in Congo discharge, but the record does not show a similar reduction in sea surface temperature. This is in marked contrast to the Cariaco Basin record from the western equatorial Atlantic, where sea surface temperatures were low throughout the Younger Dryas (6, 7). Instead of a direct control from sea surface temperatures, Weldeab *et al.* suggest that an atmospheric reorganization in the North Atlantic may have been propagated by meridional circulation systems to reduce monsoon intensity.

At the multimillennial time scale, discharge rates from the Congo Basin are found to be positively related to warmer episodes of the past 155,000 years. This result concurs well with findings from continental African sites—including Lake Bosumtwi, Ghana, a meteorite impact crater with sediments accumulated over the course of 1 million years that is the current focus of an International Continental Drilling Programme project.

The past 30,000 years from Lake Bosumtwi (8) have become a cornerstone of African paleoclimate research and offer a useful terrestrial comparator to the Congo discharge record. Reduced freshwater discharge occurred during the Last Glacial maximum, when large parts of Africa were relatively dry. Dry conditions persisted in most regions until about 15,000 years ago, when Congo discharge and the levels of many African lakes, including Bosumtwi, began to increase dramatically. DeMenocal *et al.* have hypothesized that the disproportionate intensification of monsoon precipitation after 15,000 years was triggered by a threshold response to rising insolation (9). Discharge from the Congo peaked around 10,500 years ago, at a time when Lake Bosumtwi was at its maximum and was overflowing.

More puzzling is the gradual and continuous decline in Congo discharge from the early Holocene maxima shown by the new data. This

gradual transition differs from the much more abrupt shift from wet to dry conditions about 5000 years ago shown in many lake-level curves and offshore dust records (9). Vegetation die-back as the climate dried could increase erosion potential and therefore dust supply in a steplike fashion. The more abrupt response shown by lake data might either be due to the sensitivities of the climate proxies used in the reconstructions or could indicate differences in the rate of precipitation decline between central equatorial Africa and neighboring regions.

Hydrological changes in the tropics amplify global climate changes through regulation of water vapor, change in heat transport, methane release from wetlands, and shifts in surface albedo. Spatial differences in hydrological variability will be unveiled as more sites like the Gulf of Guinea and high-resolution continental sites are investigated. There is abundant evidence of the tropics responding to changes driven from higher latitudes, but there are also suggestions from some sites that change in tropical regions may take the lead. For example, a shift toward greater aridity occurs in several African lakes 200 to 400 years before the well-known cold interval at 8200 years before present (8) found in Greenland (10) and many northern temperate

records. Similarly, well-dated continental records extending to before the Eemian interglacial (more than 135,000 years ago) are beginning to suggest a tropical lead out of glaciation through either generation of greenhouse gases from wetland expansion or strengthened meridional circulation (11).

Data of high temporal resolution from prized localities challenge the modeling community to resolve the climate dynamics behind ocean-continent interactions.

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PLANT SCIENCE

Infectious Heresy

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Not all symbiotic bacteria that fix nitrogen for legumes do so by secreting a specific nodule-initiating factor. Two strains use an alternative strategy.

Leguminous plants, such as peas and soybeans, enter into a symbiotic relationship with soil bacteria called rhizobia. For years it has been the accepted wisdom that Nodulation (Nod) factors secreted by rhizobia enable them to infect a legume and initiate formation of nodules on the host plant's roots. Within these nodules, the bacteria convert free nitrogen to ammonia, which the plant uses for its growth. On page 1307 in this issue, Giraud *et al.* (1) provide evidence that overturns this orthodoxy. They determined that the genomes of two strains of legume-nodulating rhizobia do not contain genes that are necessary for the synthesis of Nod factors. This means that these bacteria must have an alter-

native way of initiating the dialogue that results in legume nodulation.

The symbiosis between legumes and rhizobia is an agriculturally important relationship because such legumes grow well without added nitrogen fertilizer. Nod factors activate a plant signaling pathway that induces oscillations in the concentration of intracellular calcium (called calcium spiking). Calcium spiking triggers the expression of genes that are required for nodule morphogenesis in roots (2). Nodulation is intimately linked with the establishment of threadlike structures that convey rhizobia into nodule cells (see the figure).

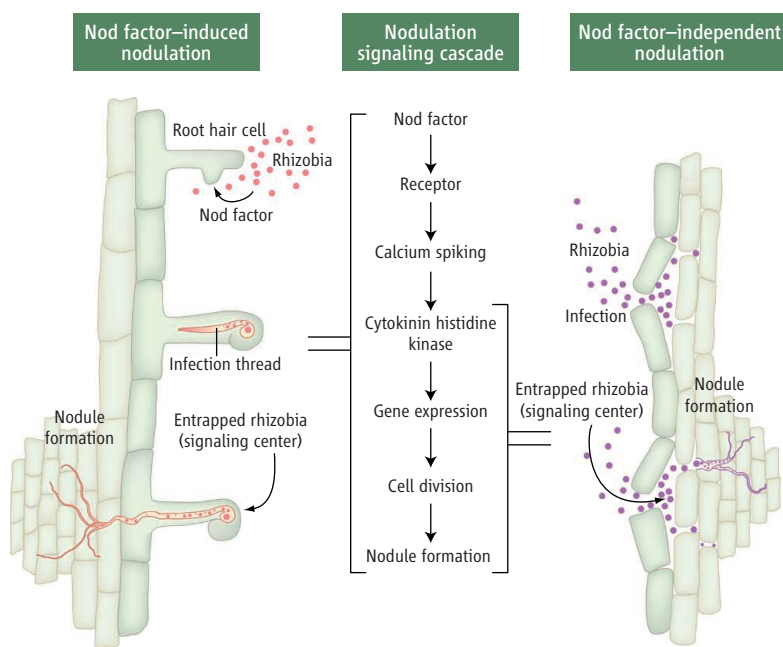
The two rhizobia (ORS278 and BTAi1) sequenced by Giraud *et al.* are unusual because they induce nodule formation on both stems and roots of some legumes. Stem nodulation can occur on some legumes that undergo periods of flooding, which enables

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colonization of stems by rhizobia. Another oddity is that these strains engage in photosynthesis and contain a cluster of photosynthesis-related genes that are similar to those found in closely related photosynthetic bacteria. However, the real surprise is that neither strain contains *nodA* or *nodC* genes, which are essential for the synthesis of Nod factors. NodC produces chitin oligomers from uridine 5'-diphosphate-*N*-acetylglucosamine, and NodA transfers a long-chain acyl group onto the chitin molecules to form a core Nod-factor structure, which can then be modified by other *nod* gene products (3).

How do ORS278 and BTAi1 infect and induce development of nitrogen-fixing nodules on their hosts, the aquatic legumes *Aeschynomene sensitiva* and *Aeschynomene indica*? Not only are these bacteria unusual, but so are these legumes. To demonstrate this, Giraud *et al.* used a *nod*-gene containing *Bradyrhizobium* strain (ORS285), which nodulates other legumes in addition to *A. sensitiva*. Mutation of the *nod* genes in ORS285 does not affect nodulation of *A. sensitiva* but does block nodulation of the other legumes. This shows that unlike the other legumes, *A. sensitiva* can respond to a rhizobial signal other than Nod factor.

How does *A. sensitiva* induce nodule development without Nod-factor signaling? An important clue comes from recent work on legume mutants that spontaneously form nodules in the absence of any bacteria or Nod factors. Constitutive activation of lotus histidine kinase 1 (LHK1), a cytokinin hormone receptor (4) that is required for nodulation (5) in the legume *Lotus japonicus*, results in spontaneous formation of nodules in the absence of rhizobia or Nod factors. This activated cytokinin receptor appears to bypass several of the early steps in the Nod factor-activated signaling cascade, including the gene products required for calcium spiking (4). Other evidence supporting a nodulation role for cytokinins is that a strain of *Sinorhizobium meliloti* that lacks Nod factor but is genetically engineered to secrete a cytokinin, can initiate nodule morphogenesis (6). Also, treatment of certain legumes with cytokinins induces the



Getting, or not getting, the Nod. Nod factors produced by symbiotic bacteria infect the legume host and initiate the formation of nitrogen-fixing nodules (left). Nod factor-independent nodulation may result from rhizobial colonization through cracks in the root epidermis (right). Increased sensitivity to a cytokinin-like signal may allow part of the normal Nod-factor signaling cascade (middle) to be bypassed, leading to nodulation and infection in the absence of Nod factors.

expression of several genes that are expressed during normal nodule development (7).

To identify the signal that ORS278 and BTAi1 use to initiate nodulation, Giraud *et al.* screened for mutants of ORS278 that could not induce nodule formation in *A. sensitiva*. Although no mutants completely defective for nodulation were found, some were greatly impaired for nodulation. Several of these mutants had defective purine biosynthesis. Because plant cytokinins are derived from the purine adenine, this suggested that the bacterial mutants might produce smaller amounts of a cytokinin (or cytokinin precursor) that initiates nodulation. Supporting a possible role for such a signal during conventional nodulation is the observation that other rhizobia carrying mutations in purine biosynthesis are defective for infection of nodules in common legumes (8). Furthermore, proteins similar to a previously unrecognized plant cytokinin-activating enzyme, encoded by the rice *LONELY GUY* gene, may be present in agrobacteria (9). However, in the absence of a mutant that is completely nodulation defective, Giraud *et al.* cannot formally distinguish between a signal that activates the entire nodulation signaling pathway or a signal that bypasses the signaling pathway by stimulating production of a cytokinin.

If Nod factors are not required for nodulation in some legumes, why should the Nod fac-

tor-induced nodulation pathway be so predominant? Formation of nitrogen-fixing nodules requires rhizobial infection, and this requires entrapment of the bacteria. During "conventional" infection, Nod factors induce root-hair deformation, leading to bacterial entrapment. Subsequent growth of infection threads requires modification of Nod factors (as specified by *nod* genes), and probably increased concentrations of Nod factors. In some legumes, however, bacteria gain entry to roots through cracks in the epidermis, often at the sites where lateral roots emerge. In another well-studied stem-nodulating legume (*Sesbania rostrata*), this type of infection shows much less specificity for Nod-factor structure, although it does require some type of Nod factor (10). The mode of infection of *A. sensitiva* and

A. indica by ORS278 and BTAi1 shows similarities to that seen in *S. rostrata*. Such entry through cracks may allow bacteria to accumulate and form a "signaling center" (10) that can induce nodulation and infection (see the figure). Nodule development could occur if a cytokinin-type signal accumulates in such a signaling center and bypasses the early Nod-factor signaling pathway. However, induction of infection-thread growth without Nod factors is highly unusual. Therefore, for both nodule and infection-thread development to occur in this system implies that *A. sensitiva* and *A. indica* may enhance intercellular colonization by bacteria and/or be unusually sensitive to some bacterially made signal. Whether this signal is a cytokinin or not remains to be established.

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