mathematical mechanism whereby the giant component emerges in an evolving network is related to the mechanism whereby a spreading disease in such a network becomes an epidemic (*3*). Perhaps there are nonclassical, but still natural, models for the spread of a disease in a network for which epidemics emerge in unexpected ways.

It is important to note that the results presented by Achlioptas *et al.* are given by computer simulation rather than formal mathematical proof. So it may be the case (although it seems unlikely) that for larger values of n, some other kind of behavior becomes apparent. Indeed, whether or not this happens is a very intriguing mathematical issue. This question will certainly draw considerable attention in the near future, and its solution (like the solution of any mathematical problem that appears to be beyond the reach of the current state of the art in mathematical technique) may lead to deeper insights into the evolution of randomized network formation models in general.

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PLANT SCIENCE

Paternal Patterning Cue

A signaling factor in sperm couples fertilization to the first plant patterning event.

Ueli Grossniklaus

More than 2000 years ago, Aristotle reflected on the contributions of mother and father to their offspring and proposed that the mother provided "matter" while the father provided "form" (*1*). The former is best illustrated by the development of enucleated sea urchin eggs into normal pluteus larvae without any contribution from the zygotic genome (*2*). In plants, it was long thought that any parental effects on embryogenesis were nonexistent. Over the past decade, however, several mutations that exert maternal effects on embryogenesis have been described in the model plant *Arabidopsis thaliana* (*3*). On page 1485 of this issue, Bayer *et al.* (*4*) describe the first paternal effect on plant embryogenesis, demonstrating that a temporal cue provided by the sperm cell triggers the events leading to the first asymmetric division of the plant embryo.

Unlike in animals, where the products of meiosis (cell division that forms gametes) differentiate directly into haploid egg and sperm (harboring one set of chromosomes), plant spores divide to form multicellular gametophytes. The male gametophyte (pollen) harbors two sperm cells, which are delivered to the female gametophyte (embryo sac) that is embedded in the ovule, the precursor of the seed. In flowering plants (such as *Arabidopsis*), one sperm fuses with the egg cell to form the zygote, whereas the second fuses with the central cell and develops into the endosperm, a nutritive tissue supporting the growth of the embryo. After fertilization, the zygote elongates and divides asymmetrically to form a small apical cell, the precursor of the embryo proper, and a large basal cell, which develops into a filamentous structure called the suspensor (see the figure). *Arabidopsis* zygotes that inherit a paternal mutant *short suspensor* (*ssp*) allele fail to elongate and show defects in suspensor development. In extreme cases, the suspensor is completely lacking, implicating *SSP* in promoting suspensor fate (*4*).

Bayer *et al.* show that in *Arabidopsis*, the *SSP* gene encodes an interleukin-1 receptor–associated kinase/Pelle-like kinase. This enzyme activates a signaling pathway in the zygote that involves the MAP kinase kinase kinase YODA (YDA) and the MAP kinases MPK3 and MPK6. This cascade of activated kinases (the *YDA* pathway) promotes elongation of the zygote and suspensor development (*5*, *6*). The SSP protein contains an amino-terminal motif for myristoylation/palmitoylation (diacylation), a central kinase domain, and a carboxyl-terminal tetratricopeptide repeat.

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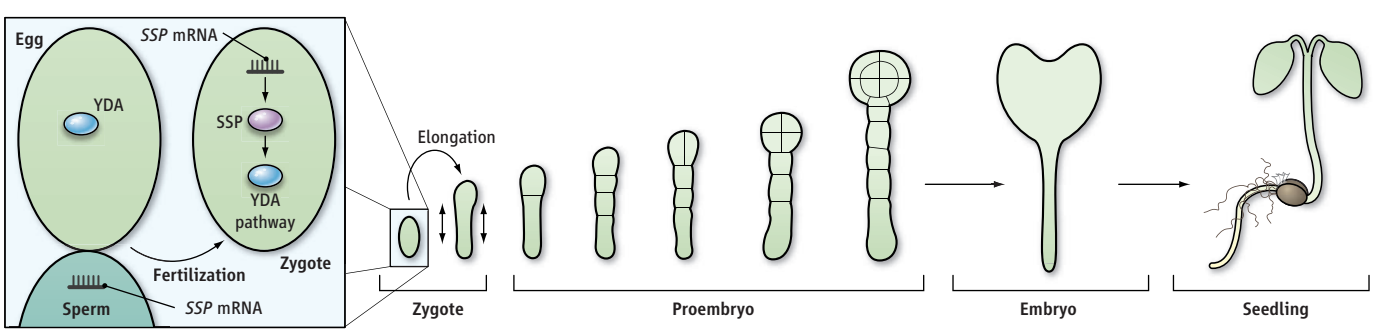

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Developmental direction. Early development of the *Arabidopsis* embryo includes zygote elongation, asymmetric cell division, and the differentiation of distinct fates. Factors that trigger zygote elongation and the first asymmetric cell division after fertilization are shown.

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Although membrane association through the diacyl modification and the repeats are essential for SSP function, the kinase domain is dispensable. These findings strongly suggest that SSP acts at the plasma membrane, possibly by recruiting a *YDA* pathway activator.

Interestingly, the *ssp* mutation affects early embryogenesis only if it is paternally inherited. Though parent-of-origin effects were already known to mule breeders in Asia Minor more than 3000 years ago (*7*), genetic parent-of origin effects were only recognized in the 1950s. In plants, which can be regenerated from single cells in culture through somatic embryogenesis, parental effects influencing embryogenesis were not thought to play a crucial role. In general, parent-of-origin effects can be mediated through nonnuclear, cytoplasmic contributions by the gametes or through the nonequivalent contribution of maternal and paternal alleles. The former is well illustrated by maternal effects in the fruit fly *Drosophila melanogaster*, where body axis determination depends on maternally stored cytoplasmic products (*8*). The latter, referred to as genomic imprinting, has only been described in mammals and seed plants. In *Arabidopsis*, both types of parent-of-origin effects likely exist. The first maternal effect gene identified in *Arabidopsis* turned out to be regulated by genomic imprinting (*9*). Furthermore, about half of the female gametophytic mutants isolated to date show defects in early seed development (*3*), and most of the paternally inherited genome is silent or active only at a low level during the first few embryonic divisions in maize and *Arabidopsis* (*10*, *11*). Both findings suggest that there may be extensive maternal control over early embryogenesis that is mediated, in part, by cytoplasmically stored products (*12*).

The mechanism by which *SSP* exerts its paternal effect may provide a means to subvert this maternal predominance. Bayer *et al.* show that mRNAs encoding *SSP* are only present in mature sperm cells, where they are apparently not translated into protein. By contrast, SSP protein is transiently detectable in the zygote and endosperm, suggesting that it is produced from paternally provided transcripts upon fusion of the egg and central cell with the sperm cells. Given the complex complement of transcripts present in plant sperm cells (*13*, *14*), more paternal effect genes may be discovered. Whether this mechanism evolved as a consequence of a parental conflict, as has been proposed for the evolution of genomic imprinting (*9*), remains to be determined.

The known mutations in *Arabidopsis* that disrupt imprinted loci show normal early embryonic development, but affect cell proliferation of embryo and endosperm at later stages and eventually lead to seed abortion. By contrast, *spp* mutants are viable and have no effect on endosperm development, but affect the very first, asymmetric division of the zygote. Thus, *SSP* transcripts delivered to the zygote by the sperm provide a molecular cue that links fertilization to the first zygotic division, which establishes apical-basal polarity of the embryo. This mechanism ensures that the activation of the *YDA* signaling pathway can only occur after fertilization and, thus, provides a temporal cue to initiate embryogenesis. Such a temporal cue would be of particular importance if most factors required for early development are already stored in the egg (*12*). This leaves the question, however, as to how embryonic activation is (de)regulated in apomictic plants, in which an egg develops into an embryo in the absence of fertilization (*15*). Is the *YDA* signaling pathway activated independently of *SSP*, or is *SSP* expressed from the maternal allele in the egg of apomictic plants? Future investigations will reveal how prominent paternal cues are to stimulate embryonic development, which predominantly depends on maternally provided factors, at least in animals and likely also in plants. In other words, how instructive is the Aristotelean paternal "form" to the maternal "matter"?

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CIRCADIAN RHYTHMS

Linking the Loops

C. Robertson McClung

A new module in the plant circadian clock provides a long-missing link in the oscillator.

The evolution of life on a rotating planet has placed a premium on the temporal coordination of biological function with dramatic daily changes in environment. Thus, organisms from cyanobacteria to humans have evolved circadian clocks, endogenous oscillators with periods approximating the solar day, which provide temporal organization of many biological processes. The circadian clocks of different taxonomic groups comprise unrelated proteins, suggesting multiple evolutionary origins. Despite this phylogenetic diversity, there is a common logic to the molecular circuitry of these clocks—they are composed of feedback loops with positive and negative components (*1*). On page 1481 of this issue, Pruneda-Paz *et al.* (*2*) solve a major puzzle in our understanding of the plant clock and provide mechanistic insight into the positive arm of a core oscillatory loop first described nearly a decade ago (*3*).

Circadian clocks are composed of multiple interlocked feedback loops (*1*). Such complexity may increase clock stability and enhance the flexibility of response to multiple exogenous and endogenous time cues, thus integrating environmental signals with metabolic and physiologic information (*4*). The resonance of the internal periodicity imposed by the endogenous circadian clock with the environmental period imposed by Earth's rotation is important for fitness. For example, net photosynthesis falls dramatically when internal and external periods diverge (*5*). Recently, it was shown that altered clock function contributes to the increased growth, called "hybrid vigor," observed in hybrids and allopolyploids (*6*).

The clock of the plant *Arabidopsis thaliana* includes at least three interlocked loops (*7*–*9*). The initial identification of a putative core clock feedback loop in plants came with the establishment of reciprocal regulation between two Myb transcription factors—CIRCADIAN CLOCK ASSOCIATED 1 (CCA1) and LATE ELONGATED HYPOCOTYL (LHY)—and a pseudo-response regulator (PRR) called TIMING OF CAB EXPRESSION 1 (TOC1) (*3*). CCA1 and LHY also participate in a second

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