The fossil record shows that prokaryotes—the cyanobacteria in particular—appeared on earth about three and a half billion years ago. This development is impressively speedy in light of the fact that it was only 3.8 billion years ago that the earth cooled sufficiently to allow solid rocks to form. Indeed, eukaryotes didn’t evolve until about one and a half billion years ago, or two billion years after the appearance of the first prokaryotes. The earliest records of multicellular life date from a little over a half billion years ago.

The protracted period between prokaryotic and multicellular eukaryotic evolution most certainly reflects the associated increase in biological complexity: larger genome and cell size; the elaboration of eukaryotic membranes, including the hallmark nuclear envelope; and the endosymbiosis of mitochondrial and plastid forerunners. Nevertheless, the specific mechanisms and chronology underlying the evolution of eukaryotic characteristics remain under debate (see Patterson, 1999). For instance, based on the identification of amitochondriate nucleate protists, the symbiotic recruitment of mitochondria and plastids has been surmised to have occurred subsequent to the elaboration of the nuclear envelope and other endomembrane structures. This conclusion, however, has been challenged more recently by the finding that amitochondriate protists appear to have internalized mitochondrial genes into the nuclear genome prior to the evolutionary discarding of the organelles. Thus, amitochondriate protists can not be regarded as the unqualified forerunners of mitochondriate eukaryotes.

In any event, an irrefutable degree of complexity in eukaryotic evolution can be seen with the appearance, one billion years after eukaryotes first appear in the fossil record, of macroscopic life. Now I do not wish to espouse teleological arguments, for I acknowledge that cellular and molecular simplicity can offer selective advantages; the unparalleled range of niches habitable by bacteria attests to this. But I would like to stress again that the biological challenges that engender multicellular life were not somehow broached and transcended by early unicellular eukaryotes subsequent to the evolution of nuclei, mitochondria, and chloroplasts. Rather, the evolutionary pathway to multicellular existence began with the very appearance of the first eukaryotic cell. What, after all, is the endosymbiotic theory of eukaryotic assembly if not the recognition of the primitive instance of multicellular life?

By definition, endosymbiosis is a cellular venture, and one that is generally regarded as obligatory on the part of both biological participants. Subsequent to a mutually obligatory association between primitive prokaryotes, then, the evolutionary development of bona fide plastid and mitochondrial organelles is envisaged to have entailed the transfer of genetic material from endosymbiont to host genome. This transfer inevitably functioned to stabilize the organelle-nucleus partnerships that characterize higher organisms. Indeed, the adoption of endosymbiont genes by the nucleus has proven in many instances to be quite extensive. As an example, only 13 protein-encoding genes persist within the genome of the human mitochondrial genome, compared to 62 for the mitochondrial genome of the primitive heterotrophic flagellate, Reclinomonas americana (Lang et al., 1997; see also Palmer, 1997). At the same time, the very fact that mitochondria have been maintained with their own genomes by all multicellular organisms, must not be overlooked. Indeed, the Arabidopsis mitochondrial genome is much larger than the human mitochondrial genome and contains 27 protein-encoding genes, which should by no means be interpreted to signify greater independence from the nucleus as compared to the case in human cells. (And as shown by Turmel et al. on pages 1717–1729 and by Burger et al. on pages 1675–1694 in this issue, the number and arrangement of mitochondrial genes can vary even within a single eukaryotic lineage.)

As a consequence of the intranuclear localization of genes essential to mitochondrial and plastid metabolism, mechanisms for the routing of nuclear-encoded gene products from the cytoplasm into the given organelles became necessary (for reviews of these mechanisms within plant cells, see Glaser et al., 1998; Keegstra and Cline, 1999). The need to traffic macromolecules, in turn, can be viewed as an extension of (or a preface to) the same demands placed on eukaryotes by virtue of their enveloped nucleus and endomembrane system. Specifically, information stored within the nucleus must be intermittently relayed across membranes and must additionally be made accessible to regulatory cues that are orchestrated so as to penetrate into the nucleus. Thus, multicellular forms of life, which must negotiate the exigencies of intercellular communication and cellular specialization, might well be viewed as the inevitable predicate to demands of information flow that were first orchestrated intracellularly between organelles.

The concerted elaboration of cellular constellations, tissues, and organs in higher organisms has been intensively
studied for the last half-century, and the bases of cell-cell communication that underlie developmental processes comprise entire branches of biology. Studies into the interorganellar communication that supports eukaryotic life have somewhat lagged intercellular considerations, despite the mechanistic and evolutionary heritages that the two levels of biomolecular communication share. Just how common the mechanisms of interorganellar and intercellular communication will prove to be, however, is not clear.

Processes of interorganellar communication within plant cells will be characterized by several dimensions not typical of animal cells. Specifically, avenues of plastid-nucleus as well as plastid-mitochondrion communication must be established in the plant cell, in addition to the mitochondrion-nucleus interactions represented within non-plant eukaryotes. As with virtually all other metabolic processes in plant cells, light plays an important regulatory role in these interactions. Indeed, several mutations have been imposed upon plant systems that confirm the importance of each of these dimensions to proper development (see Chory and Suske, 1994).

In this issue of THE PLANT CELL, three articles address the coordination of nuclear-organellar metabolism. In one, on pages 1799–1810, Kapoor and Sugiura identify two sequences within the promoters of plastid-encoded genes that are recognized and transcribed by a specific nucleus-encoded RNA polymerase from tobacco. In a second, on pages 1709–1716, McCormac and Barkan investigate not the transcription, but the translation of the maize chloroplast atpB/E dicistronic mRNA and the relevance of a nuclear locus in promoting proper translation within the organelle. The nuclear mutation, denoted atp1, specifically affects translation, having no effect on either the accumulation or structure of the atpB/E message, and thereby results in the obliteration of the chloroplast ATP synthase complex. The effect of atp1 on chloroplast metabolism bespeaks the capacity for nuclear-encoded information to regulate specific organellar functions. This type of nuclear influence on specific organellar translation has been described in only one previous instance (i.e., crp1 in maize; Barkan et al., 1994). It is not clear how the mutation of the nuclear atp1 locus exerts its effect on translation of the specific chloroplast mRNA. Nevertheless, both articles represent specific examples of how a nuclear genome encodes information that is brought to bear upon another genome (i.e., a plastid genome).

As outlined previously, such nucleus-to-organelle communication can be taken as the defining characteristic of eukaryotic life. But information flow between organelles must often be bidirectional, an exigency that is illustrated in the third article that addresses interorganellar communication in this issue. On pages 1609–1621, Streathfield et al. demonstrate that defects in plastid metabolism—in this instance arising from a mutation in a nucleus-encoded plastid protein—can culminate in the altered expression of specific nuclear genes. The authors' work extends upon previous analysis of the Arabidopsis cue1 (for chlorophyll a/b binding protein [CAB] underexpressed) mutant, isolated for its limited expression of the light-regulated nuclear CAB gene. Mutations in cue1 are phenotypically manifested in the form of underdeveloped mesophyll cell chloroplasts in interveinal tissue (Li et al., 1995). Like the maize atp1 locus and the nucleus-encoded plastid RNA polymerase discussed above, the CAB genes represent plastid functions that are nuclear-encoded. CUE1, although likewise a nuclear-encoded plastid factor, additionally exemplifies the potential of organellar factors to affect nuclear metabolism. The nature of this effect is now clarified in that the authors show here that CUE1 encodes a plastid inner membrane phosphate transloca-

tor that supplies the plastid stroma with phosphoenolpyruvate (PEP).

The metabolism of PEP within the chloroplast is profoundly important to plant life. The shikimate pathway, leading to the synthesis of the three aromatic amino acids, initiates within the chloroplast upon the condensation of PEP with erythrose-4-phosphate. Secondary metabolites such as lignin, pigments, UV light protectants, and phenolic redox molecules are additionally elaborated as molecular postscripts to the shikimate pathway (for review, see Herrmann, 1995). In light of the pertinent biosynthetic processes, it can come as no surprise that cue1, as a genetic lesion to the primary means for translocating PEP into the chloroplast, proves to be pleiotropic. Among the phenotypic consequences of cue1 (besides reduced CAB message) are a perturbation in plastid redox poise and decreased photosynthetic efficiency, reduced cellular content of aromatic compounds, significantly elevated amino acid and ammonia levels, decreased biomass accumulation, and reticulate leaf morphology. (This latter trait was in fact useful in isolating multiple alleles of cue1.) Administration of Phe, Tyr, and Trp can ameliorate the phenotypic consequences of the mutation, which suggests that the fundamental role of the PEP/phosphate translocator (PPT) is indeed to feed substrate into the shikimate pathway.

Given the far-reaching, gross insults to cellular metabolism that are engendered by cue1, then, it might well be argued that specific avenues of interorganellar communication cannot be directly assessed. The pleiotropy of the cue1 mutation is indeed an experimental challenge, but may in itself represent the importance of proper interorganellar communication to the maintenance of intracellular homeostasis. Moreover, the various cue1-conferring traits are sensitive to light intensity, just as would be expected from perturbations of light-regulated interorganellar lines of
communication. Finally, it must also be reckoned that the influence of light intensity upon cue1 mutants, along with the fact that the mutation fundamentally affects the anabolism of aromatic photoprotectant compounds, arises from photooxidative damage. But again, it makes good biological sense that photooxidative byproducts would inform plastid signaling to the nucleus.

As intercellular communication in plant systems continues to be experimentally elaborated in terms of signal transduction and reaction cascades that ultimately relay information to the nucleus, it may seem counterintuitive that a “housekeeping” protein (e.g., PPT or, equivalently, CUE1) in the plastid inner membrane would contribute to intracellular pathways of communication. The organellar complexity of plant cells in particular nevertheless calls for an investigation of such aspects of our eukaryotic heritage. Clearly, further characterization of CUE1 is necessary before its role in interorganellar signaling can be delineated—indeed, Streatfield et al. indicate that they are engaged in studies of how cue1 influences regulation of CAB.

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