For quantum weirdness with more kick to it, we need look no further than the two delayed-choice experiments of Kaiser *et al*. and Peruzzo *et al*. Both experiments use quantum entanglement to delay the choice of what quantum effects are demonstrated not merely until after the photon has entered the interferometer, but until after the photon has emerged from the interferometer and the measurement that detects it has already taken place. In the first proquastination experiment, polarizing beam splitters ensure that vertically polarized photons entering the Mach-Zehnder interferometer undergo quantum interference, while horizontally polarized photons do not. Photons whose polarization is in between vertical and horizontal—diagonally polarized photons—exhibit partial interference.

There is nothing here that the two Ludwigs, Mach and Zehnder, couldn't already have observed in the early 1890s, but now the tricky part comes in. Kaiser *et al*. do not send a photon with a definite polarization into the interferometer. Rather, they send a photon whose polarization is entangled with the polarization of a second photon. After the first photon has already emerged from the interferometer and the port by which it has emerged has been detected, Kaiser *et al*. measured the polarization of the second photon. If they measure the polarization of the second photon along the vertical/horizontal axis and obtain the result "horizontal," then the first photon has behaved like a particle: No interference has taken place. If they obtain the result "vertical," then the first particle has behaved like a wave, and interference has taken place.

So far, the results of the experiment could be explained simply by saying the two photons are either both horizontally polarized or both vertically polarized. If one chooses to measure the second photon along the diagonal/antidiagonal axis however, so that first photon exhibits partial interference, then Bell's inequalities (*9*) can be used to show that this convenient classical explanation won't wash. It is the measurement on the second photon—apparently retroactively—that made interference take place or not.

The second demonstration of quantum procrastination, by Peruzzo *et al*., is if anything even more audacious. In this experiment, a photon is sent through a Mach-Zehnder interferometer as before, but the presence or absence of the second beam splitter in the Mach-Zehnder interferometer is entangled with the state of a second photon. As a result, even after the first photon has been detected, the question of whether it has exhibited wave nature, particle nature, or something in between, is determined by measurements made on the second photon. Strong violations of Bell's inequalities again rule out easy classical explanation.

Although the two quantum procrastination experiments reported here delay the choice of whether to exhibit wave- or particle-like nature of entangled particles for just a few nanoseconds, if one has access to quantum memory in which to store the entanglement, the decision could be put off until tomorrow (or for as long as the memory works reliably). So why decide now? Just let those quanta slide! Sadly, the applications of quantum procrastination are for the moment limited to making only a few highly quantum types of decision ex post facto. I wish I had decided to start writing this article a week before it was due, but no amount of entanglement can hide that I decided to the day before.

References

1. F. Kaiser *et al*., *Science* **338**, 637 (2012).
2. A. Peruzzo, P. Shadbolt, N. Brunner, S. Popescu, J. L. O'Brien, *Science* **338**, 634 (2012).
3. J. A. Wheeler, in *Quantum Theory and Measurement*, J. A. Wheeler, W. H. Zurek, Eds. (Princeton Univ. Press, Princeton, NJ, 1984), pp. 182–213.
4. L. Zehnder, *Z. Instrumentenkunde* **11**, 275 (1891).
5. L. Mach, *Z. Instrumentenkunde* **12**, 89 (1891).
6. V. Jacques *et al*., *Science* **315**, 966 (2007).
7. M. A. Nielsen, I. L. Chuang, *Quantum Computation and Quantum Information* (Cambridge Univ. Press, Cambridge, UK, 10th anniversary ed., 2011).
8. Y.-H. Kim, R. Yu, S. P. Kulik, Y. Shih, M. O. Scully, *Phys. Rev. Lett.* **84**, 1 (2000).
9. J. S. Bell, *Physics* **1**, 3 (1964

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PLANT SCIENCE

Chloroplast Delivery by UPS

Identification of a membrane-anchored E3 ligase in plants reveals a role for the ubiquitin proteasome system in chloroplast development.

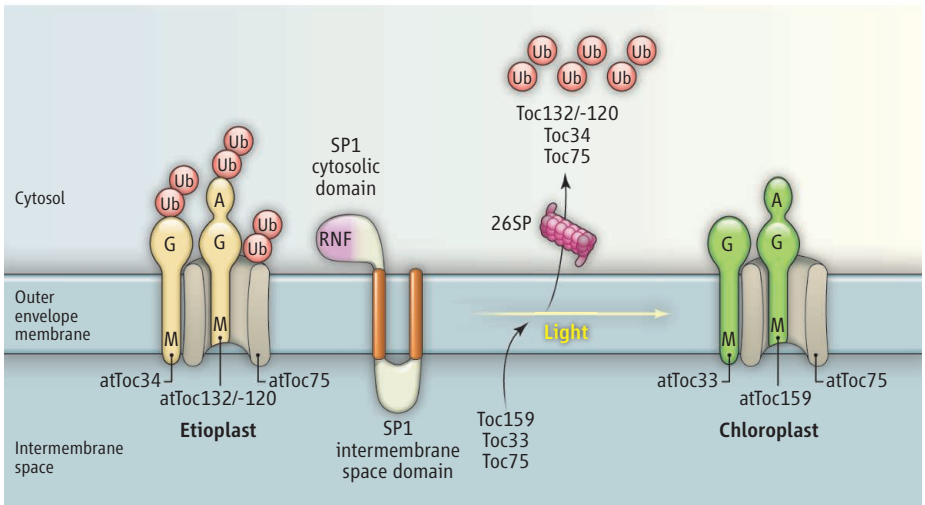
Felix Kessler

Chloroplasts are the organelles of photosynthesis in plants and are responsible for much of the food and biomass production on our planet. But chloroplasts are only the best-known members of an extended family of organelles termed plastids. Their name suggests plasticity and, indeed, plastids exist in various incarnations depending on developmental cues (e.g., nonphotosynthetic etioplasts in dark-grown leaves, colored chromoplasts in petals and fruit, and starch-storing amyloplasts in roots). Yet, the mechanisms underlying the transformation from one plastid type to another are largely unknown. On page 655 in this issue, Ling *et al*. (*1*) show that the ubiquitin-26*S* proteasome system (UPS) directly targets plastids and promotes chloroplast biogenesis, controlling yet another important facet of cell biology.

Plastids originate from an endosymbiotic process that started ~1.5 billion years ago when a eukaryotic host cell engulfed a photosynthetic prokaryote. Over time, the two organisms became almost completely integrated. A permanent and ongoing flow of genetic material from the prokaryotic endosymbiont resulted in the transfer of most plastid protein-encoding genes to the host nucleus (*2*). The *Arabidopsis* chloroplast today has ~2000 proteins (*3*, *4*), only 87 of which are encoded in the organelle. Concurrently with their transfer to the nucleus, the former endosymbiont genes acquired genetic information encoding amino-terminal targeting sequences resulting in synthesis of preproteins in the cytosol. The amino-terminal sequences enable the recognition and the translocation of preproteins across the dual-membrane chloroplast envelope and are later removed.

Preprotein recognition and envelope translocation are facilitated by the chloroplast protein import machinery (*5*), which consists of translocon complexes at the outer (TOC) and inner envelope membranes of the chloroplast. The main components (identified by their molecular mass in kilodaltons) of the Toc complex (Toc159, Toc34, and Toc75) were first identified in isolated pea chloroplasts (*6*–*8*) and play essential roles in chloroplast biogenesis in *Arabidopsis thaliana* (*9*, *10*). Toc159 and Toc34 are outer membrane preprotein receptors shar-

Institute of Biology, University of Neuchâtel, CH-2000 Neuchâtel, Switzerland. E-mail: felix.kessler@unine.ch



Model of chloroplast biogenesis. Nonphotosynthetic, undifferentiated plastids in the dark (etioplasts) import housekeeping proteins via a translocon at the outer membrane of the chloroplast (Toc) complex consisting of the preprotein receptors Toc132/-120 and Toc34 together with the protein-translocating channel Toc75. Light triggers the transformation of etioplasts into chloroplasts. Developing chloroplasts import large quantities of photosynthesis-associated proteins using a Toc complex consisting of the preprotein receptors Toc159 and Toc33 together with Toc75. The remodeling of the Toc complex during chloroplast biogenesis implicates an outer membrane E3 ligase, SP1, that targets components of the protein import machinery for degradation by the 26*S* proteasome (26SP). A, acidic; at, *Arabidopis thaliana*; G, GTP-binding; M, membrane insertion; RNF, RING-finger domain; Ub, ubiquitin.

ing homology in their guanosine 5′-triphosphate (GTP)–binding domains, and they extend into the cytosol. Toc75 forms the protein-conducting channel, deeply buried in the outer membrane. The *Arabidopsis* genome revealed that a small gene family of Toc GTPases (Toc159, -132, -120, -90, -34, and -33) engage in separate, preprotein-specific pathways (*11*, *12*). Toc159 and -33 are present predominantly in the chloroplast and mediate the import of the highly abundant proteins associated with photosynthesis. By contrast, Toc132/-120 and Toc34 are present mostly in other plastid types and are required for the import of housekeeping proteins.

Chloroplast biogenesis occurs when a dark-grown (etiolated) plant senses the light and consists of a series of developmental processes called photomorphogenesis (*13*). This results in extensive changes in gene expression that dramatically increase the components of the photosynthetic machinery and enables the greening of young plants. Greening directly reflects chloroplast biogenesis and leads to remodeling of the import machinery and then of the entire chloroplast proteome that becomes dominated by highly abundant photosynthesis-associated proteins.

For the import machinery, the balance is shifted from Toc120/-132 and Toc34 to Toc159 and Toc33. Both *toc33* (*ppi1*) (*9*) and *toc159* (*ppi2*) (*10*) mutants give rise to chloroplast phenotypes (pale green and albino, respectively), emphasizing the relevance of these components and their specific role in preprotein import. But what brings about this switch of components in the import machinery during chloroplast biogenesis?

In a screen for second site suppressors of *ppi1*, Ling *et al*. discovered SP1 (suppressor of *ppi1* locus 1), a "really interesting new gene" (RING)–type ubiquitin E3 ligase. Together with E2, E1, and the proteasome, E3 ligases regulate protein degradation and thereby a range of processes in animals and plants, and occupy 6% of the *Arabidopsis* genome (*14*). SP1 has a "really interesting" topology: It is anchored in the plastid outer envelope membrane by two transmembrane helices and exposes a carboxyl-terminal C3HC4-type RING-finger domain to the cytosol. The domain between the two transmembrane helices faces the envelope intermembrane space and interacts with its TOC targets. Thus, SP1 is ideally positioned to ubiquitinate targets at the chloroplast surface.

The *sp1 ppi1* double-mutant plants generated by Ling *et al*. are larger and greener and contain more extensively developed chloroplasts than *ppi1* but still less so than wild-type plants. By contrast, the *sp1* single mutant showed a slower transformation of etioplasts to chloroplasts, and from chloroplasts to gerontoplasts (aging chloroplasts in older plants). This indicates a role in the transformation of one plastid type to another (see the figure). In vitro and in vivo experiments by the authors show that Toc complex components are directly ubiquitinated by the action of SP1. But how could loss of ubiquitination activity by the *sp1* mutations rescue the pale phenotype and the defective chloroplasts in the *ppi1* mutant? The answer may lie in the reduced degradation and consequent increase in amounts of other Toc components that may compensate for the absence of Toc33 in the *ppi1 sp1* double mutant.

Why would the loss of SP1 function disrupt transition between different plastid types? Chloroplast biogenesis, as well as the differentiation of other plastid types, is linked to the import of functionally specific proteins that in turn require specific combinations of the import receptor homologs (i.e., Toc159 and Toc33 for photosynthesis-associated proteins and Toc132/-120 and Toc34 for housekeeping proteins). SP1-dependent ubiquitination followed by proteasome-mediated degradation may therefore allow modulation of the composition of the Toc complex with regard to the preprotein demands of the developing plastid type.

Are the Toc components the only targets of SP1, or are there other substrates? SP1 has a close homolog, SPL1, that also localizes to the chloroplast outer membrane. Genetic evidence indicates that SPL1 is not redundant with SP1, but its function remains unknown. The findings of Ling *et al*. have opened an exciting new door to regulatory mechanisms at the chloroplast import machinery. No doubt, many interesting secrets remain to be discovered.

References and Notes

1. Q. Ling, W. Huang, A. Baldwin, P. Jarvis, *Science* **338**, 655 (2012).
2. J. N. Timmis, M. A. Ayliffe, C. Y. Huang, W. Martin, *Nat. Rev. Genet.* **5**, 123 (2004).
3. B. Zybailov *et al.*, *PLoS One* **3**, e1994 (2008).
4. K. Baerenfaller *et al.*, *Science* **320**, 938 (2008).
5. F. Kessler, D. Schnell, *Curr. Opin. Cell Biol.* **21**, 494 (2009).
6. S. E. Perry, K. Keegstra, *Plant Cell* **6**, 93 (1994).
7. D. J. Schnell, F. Kessler, G. Blobel, *Science* **266**, 1007 (1994).
8. S. Hirsch, E. Muckel, F. Heemeyer, G. von Heijne, J. Soll, *Science* **266**, 1989 (1994).
9. P. Jarvis *et al.*, *Science* **282**, 100 (1998).
10. J. Bauer *et al.*, *Nature* **403**, 203 (2000).
11. S. Kubis *et al.*, *Plant Cell* **16**, 2059 (2004).
12. Y. Ivanova, M. D. Smith, K. Chen, D. J. Schnell, *Mol. Biol. Cell* **15**, 3379 (2004).
13. C. Kami, S. Lorrain, P. Hornitschek, C. Fankhauser, *Curr. Top. Dev. Biol.* **91**, 29 (2010).
14. R. D. Vierstra, *Nat. Rev. Mol. Cell Biol.* **10**, 385 (2009).

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