

Highlight: The Mystery of Mitochondrial Genomes

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Alan Christensen was drawn to an intellectual puzzle—a paradox in need of an explanation. Higher plants have bizarre mitochondrial genomes. Like nuclear genomes, these have coding and noncoding sections. When researchers compare one flowering plant with another, the coding regions are remarkably similar. The noncoding regions, however, are often so expanded, scrambled, and rearranged that they are incomparable and unrecognizable.

“The dogma is that mitochondrial genomes evolve slowly,” says Christensen, a professor at the University of Nebraska, “but that is based only on the conserved sequences that all mitochondria have. The stuff between those regions is so different that you can’t even line it up to compare it. I started to wonder, what is happening?”

Christensen proposes one possibility in a new article in *Genome Biology and Evolution* (Christensen 2013): a difference in how the DNA repair machinery behaves on transcribed and nontranscribed regions of the genomes. The repair of genes coding for mitochondrial function is very accurate, but another process, he believes, is occurring in other regions of the genome. In nontranscribed regions the process creates many duplications and expansions with no deletions, causing mitochondria to accumulate large sections of nonfunctional DNA.

“This is the first work to actually propose a mechanism to explain this pattern,” Christensen says. “Past work has been mostly concerned with describing and documenting the weirdness of plant mitochondrial genomes.”

For evolutionary biologist Douglas Taylor at the University of Virginia, who was not involved in the work, the article was a compelling, surprising read.

“I liked that Christensen outlined clear molecular pathways that might be responsible for the patterns I and others have seen,” says Taylor. Taylor has published on plant mitochondrial genomes in the past and describes his own work as “akin to natural history.” He expects Christensen’s work to influence his future research. “It might direct me toward the subsets of genes that are responsible,” he says.

To illustrate “the weirdness” of plant mitochondrial genomes, Christensen uses an analogy: Picture a series of libraries that each begin with the same collection of 100 classic

books (the genes). Each library user copies their favorite chapters from their favorite books, binds them together, and re-shelves them at random. The users are pretty bad at copying and introduce many mistakes, even recopying copies that have previously been made. The 100 classics remain intact, and are the only thing the libraries have in common with each other. Over time the copied books expand, get scrambled, and eventually become gibberish, with no relation to the original texts.

“What I asked was, where did all the junk come from, and why isn’t it as accurately maintained as the genes?” Christensen says. “By comparing two plants of the same species that were very closely related I was able to look at the processes acting on the genes and the junk both, and catch one of the sloppy copying events in action.”

Comparing two ecotypes of *Arabidopsis thaliana*, a member of the mustard family used as a model organism in plant biology, allowed Christensen to align the nontranscribed regions. Comparing the differences between both the coding and noncoding regions of the two ecotypes allowed Christensen to notice significant differences.

When both DNA strands break and are repaired, breaks in gene coding regions seem biased toward gene conversion, a form of genetic recombination that minimizes mutation rates. Double strand breaks in noncoding regions, on the other hand, seem biased toward break-induced replication. This process expands and rearranges, but does not remove, material.

“The end result over evolutionary time scales,” says Christensen, “is the plant mitochondrial genomes we see: genes in no particular order, surrounded by junk.”

Sixty percent of the two *Arabidopsis* mitochondrial genomes aligned nicely with each other, “but they’re unrecognizable outside that small plant family,” Christensen says. “So I would argue they’re actually junk.”

The low mutation rates seen in plant mitochondria have been a conundrum within the field for some time. One explanation, proposed by Michael Lynch of Indiana University, is the mutational-burden hypothesis. This suggests that low mutation rates correlate with big genomes, since the organism is at a reduced risk of a debilitating mutation. But the low mutation

rates of genes, writes Christensen, must be contrasted with the high mutation and rearrangement rates of noncoding sequences. A failure of a subset of DNA repair mechanisms, he believes, could also explain the exceptional plants with high mutation rates and extremely large genomes. If that is true, then a core molecular process is driving mitochondrial genome size, rather than mutation rate via natural selection.

Christensen suspects his proposed mechanism would hold true across the higher-order plants, but at the moment it is difficult to test his model. There are many experiments he'd like to do, but he is currently lacking a method for transforming mitochondria. (He is, however, working on developing a method for this through a project funded by the National Science Foundation.)

Taking a broader picture, Christensen's work may one day assist plant breeders. Hybrid seeds are prized for their vigor and productivity. If breeders could easily sterilize one plant sex, it would be a boon to seed production. Interestingly, when male plants are sterile it's often because of some novel mitochondrial, maternally inherited gene—something made of

patched-together other pieces of mitochondrial genome. Occasionally a nuclear gene will appear to restore fertility to the plant.

"Mitochondrial genomes influence the life history of plants. If you wanted to make hybrids, it would be nice if the female parent didn't make pollen," says Christensen. "That's the big picture of why many people are interested in plant mitochondrial genomes."

For Taylor the interest goes beyond the seed bed. "These genomes are under-appreciated. They're so complex and unusual, anything that explains them should be of general interest."

Literature Cited

Christensen A. 2013. Plant mitochondrial genome evolution can be explained by DNA repair mechanisms. *Genome Biol Evol.*, Advance Access published April 3, 2013, doi: 10.1093/gbe/evt069

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