Mitochondrial DNA, August 2012; 23(4): 321–322

informa
healthcare

MITO COMMUNICATION

Mito-communications

SIMON Y. W. HO[1] & ROBERT LANFEAR[2]

[1]*School of Biological Sciences, University of Sydney, Sydney, Australia, and* [2]*Research School of Biology, Centre for Macroevolution and Macroecology, Australian National University, Canberra, Australia*

(Received 21 March 2012; accepted 3 April 2012)

Calibrating mitochondrial rates in marine invertebrates

Rates of mitochondrial evolution vary among invertebrate lineages (Thomas et al. 2006). It also appears that estimates of mitochondrial substitution rates in invertebrates are time dependent, with higher rates being estimated in studies of short timescales (Papadopoulou et al. 2010; Ho et al. 2011). Reliable quantification of this pattern has been elusive, mainly because of the difficulty in identifying accurate sources of information for calibrating the molecular clock.

Calibrating estimates of mitochondrial rates on short timescales is a problem besetting many studies in this field. Most researchers rely on information from the fossil record or attempt to correlate the divergence of populations or species with dated biogeographic events. For example, Papadopoulou et al. (2010) used a calibration based on the formation of the mid-Aegean trench. By assuming that this geological event led to divergences between lineages of beetles, the authors were able to estimate rates of mitochondrial evolution.

In a recent study of marine invertebrates, Crandall et al. (2012) have taken a different approach to estimating mitochondrial substitution rates. They modelled the increase of available habitat and associated this with estimated population expansion. The authors estimated that a 12-fold increase in population size occurred between 14.6 and 19.6 thousand years ago on the Sunda Shelf, southeast Asia.

Estimates of substitution rates in the mitochondrial *CO1* gene ranged from 2.3×10^{-8} to 8.9×10^{-8} substitutions/site/year, depending on the species and on the date assumed for the Sunda Shelf calibration. This is considerably higher than the 'standard' arthropod rate of 1.15×10^{-8} substitutions/site/year (Brower 1994) that is widely cited in studies of invertebrates.

As with most studies of mitochondrial evolution over short timescales, there is considerable uncertainty in the rate estimates. The trend observed by Crandall et al. (2012) relies on a relatively small number of data points. By identifying reliable calibrations for recent evolutionary timeframes, we will be able to quantify rate variation in invertebrates and other taxa. This will shed light on the magnitude and causes of time-dependent rates.

References

Brower AVZ. 1994. Rapid morphological radiation and convergence among races of the butterfly *Heliconius erato* inferred from patterns of mitochondrial DNA evolution. Proc Natl Acad Sci USA 91:6491–6495.

Crandall ED, Sbrocco EJ, DeBoer TS, Barber PH, Carpenter KE. 2012. Expansion dating: Calibrating molecular clocks in marine species from expansions onto the Sunda Shelf following the last glacial maximum. Mol Biol Evol 29:707–719.

Ho SYW, Lanfear R, Bromham L, Phillips MJ, Soubrier J, Rodrigo AG, Cooper A. 2011. Time-dependent rates of molecular evolution. Mol Ecol 20:3087–3101.

Papadopoulou A, Anastasiou I, Vogler AP. 2010. Revisiting the insect mitochondrial molecular clock: The mid-Aegean trench calibration. Mol Biol Evol 27:1659–1672.

Thomas JA, Welch JJ, Woolfit M, Bromham L. 2006. There is no universal molecular clock for invertebrates, but rate variation does not scale with body size. Proc Natl Acad Sci USA 103: 7366–7371.

Correspondence: R. Lanfear, Research School of Biology, Centre for Macroevolution and Macroecology, Australian National University, Building 116 Daley Road, Canberra, Australian Capital Territory 0200, Australia. Fax: + 61 2 6125 4321. E-mail: rob.lanfear@anu.edu.au

ISSN 1940-1736 print/ISSN 1940-1744 online © 2012 Informa UK, Ltd.
DOI: 10.3109/19401736.2012.683187

Untangling the evolutionary processes that shape plant mitochondrial genomes

Plant and animal mitochondrial genomes are starkly different, and a recent study in the model plant *Arabidopsis* sheds new light on how these differences might have evolved. In animals, mitochondrial genomes are compact, non-recombining, structurally conserved and have high substitution rates. In plants, on the other hand, mitochondrial genomes vary dramatically in size, recombine frequently, show little structural conservation among species and have remarkably low substitution rates. Quite why these differences exist has puzzled even the most prominent thinkers on genome evolution (Lynch 2007), but a new study might have an important part of the answer.

Plant mitochondrial genomes frequently experience harmful DNA lesions known as double-strand breaks (DSBs). To repair DSBs, plants often use a process known as homologous recombination, which involves using an intact copy of the genome to repair the damaged copy, after matching up the two copies based on sequence similarity (Maréchal and Brisson 2010). However, plant mitochondria tend to be rich in repeated sequences. These can masquerade as homologous genome regions and mislead homologous recombination with potentially disastrous consequences. To cope with this, plants have evolved elaborate surveillance mechanisms to regulate homologous recombination in their mitochondrial genomes (Maréchal and Brisson 2010).

Davila et al. (2011) studied mitochondrial recombination using a mutant strain of *Arabidopsis thaliana* with a defective mitochondrial surveillance system. As expected, these plants had an elevated rate of homologous recombination among repeat sequences, and the researchers analysed these sequences to gain a better understanding of the underlying mechanics of homologous recombination. Davila et al. observed that during recombination between non-identical repeat sequences, nucleotide mismatches between repeats were eliminated by mismatch repair – a process known as gene conversion. Furthermore, a detailed analysis of the mitochondrial genomes of three different ecotypes suggested that a great deal of the observed variation in these genomes could be attributed to the repair of DSBs. Finally, Davilia et al. assembled and compared mitochondrial sequences from 72 natural *Arabidopsis* ecotypes. What they found was striking. First, patterns of nucleotide diversity corresponded very strongly with patterns of genome rearrangement among ecotypes, exactly as expected if DSB repair is central to plant mitochondrial genome evolution. Second, the repeat regions that underwent the most DSB repair also had the lowest levels of observed polymorphism, highlighting the importance of gene conversion in homogenising sequences.

The upshot of all of this is that DSB repair seems to be a major player in plant mitochondrial genome evolution. But what does this have to do with the difference between animal and plant mitochondria? As it turns out, it might be the missing piece of the long-studied puzzle (Galtier 2011). First, DSB repair is a potent source of genome rearrangement, because it often involves recombination between repeat regions within the mitochondrial genome. So, the prevalence of DSB repair in plants might explain why the structure of plant mitochondrial genomes evolves more quickly than that of animals. Second, Davila et al. showed that DSB repair involves gene conversion in the plant mitochondrial genome. Gene conversion tends to eliminate new point mutations, and so DSB repair might explain why the substitution rate of plant mitochondrial genomes is so much lower than that of animals. Further evidence for these hypotheses comes from a striking natural experiment – the mitochondrial genomes of corals and sponges contain mitochondrial recombination surveillance genes similar to those in plants (Abdelnoor et al. 2006), and like plants they also have notably low substitution rates (Hellberg 2006). This is unlikely to be mere coincidence, and has the satisfying feeling of pieces of the evolutionary puzzle falling neatly into place.

References

Abdelnoor RV, Christensen AC, Mohammed S, Munoz-Castillo B, Moriyama H, Mackenzie SA. 2006. Mitochondrial genome dynamics in plants and animals: Convergent gene fusions of a MutS homologue. J Mol Evol 63:165–173.

Davila JI, Arrieta-Montiel MP, Wamboldt Y, Cao J, Hagmann J, Shedge V, Xu Y-Z, Weigel D, Mackenzie SA. 2011. Double-strand break repair processes drive evolution of the mitochondrial genome in Arabidopsis. BMC Biol 9:64.

Galtier N. 2011. The intriguing evolutionary dynamics of plant mitochondrial DNA. BMC Biol 9:61.

Hellberg ME. 2006. No variation and low synonymous substitution rates in coral mtDNA despite high nuclear variation. BMC Evol Biol 6:24.

Lynch M. 2007. The origins of genome architecture. 1st ed., Sinauer Associates.

Maréchal A, Brisson N. 2010. Recombination and the maintenance of plant organelle genome stability. New Phytol 186:299–317.