

## A True Symbiosis for the Mitochondria Evolution

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### Introduction

Endosymbiotic theory (or Symbiogenesis) is an evolutionary theory that was initially proposed more than 100 years ago [1] to explain the origins of eukaryotic cells from the prokaryotic ones. This theory postulated that several key organelles of eukaryotes could have been originated as a symbiosis between separate organisms. Later in the 1920s, this idea was extended to explain the origins of mitochondria [2] and, some 30 years ago, it was definitively accepted [3]. According to this theory, a pro-bacteria was hosted by an eukaryotic cell and evolved within it, providing the host with all the ATP that is needed (therefore acting as a cellular “power house”). Some other studies were confirming this hypothesis by assessing that substantial gene additions were impossible without the energetic boost provided by the colonizing mitochondrion in the eukaryotic lineage [4].

More recently [5] this idea was questioned by a work in which the authors analyzed the cellular energetics and genomics data from a wide variety of species obtaining an indication that, relatively to the ATP requirements of a cell throughout its life, the costs of a gene at the DNA, RNA, and protein levels decline with cell volume in both bacteria and eukaryotes. These costs are usually sufficiently large to be perceived by natural selection in bacterial populations, but not in eukaryotes experiencing high levels of random genetic drift. Therefore, probably due to scaling reasons that are not yet understood, in virtue of their large size eukaryotic cells are subject to a broader set of opportunities for the colonization of novel genes which can show weakly advantageous or even transiently disadvantageous phenotypic effects. These results indicate that the origin of the mitochondrion is not mandatory for genome-size expansion.

During the last couple of years, all the above mentioned observations gave rise to an argument between Lynch and Marinov [6] and Lane and Martin [7] while, at the same time independent groups, were discovering several new findings on mitochondrial origins. Especially important was the discovery of a phylum of the Archaea, the Lokiarchaeota. This new phylum of the Archaea displays some characteristics of Archaea and some of Eukaryota: its genome indeed encodes many eukaryote-specific features. Lokiarchaeota has a great development of internal membranes and this characteristic is also due to a high presence of small G-proteins of the Rab family [8] and actin. This last protein is essential for phagocytosis in eukaryotes, and possessing such ability would facilitate the endosymbiotic origin of mitochondria, making the “leap” from prokaryotes to eukaryotes possible. Another recent work reports the first example of a eukaryote lacking any form of mitochondrion, demonstrating that this organelle is absolutely not essential for the viability of a eukaryotic cell [9] and another group was assessing that, probably, mitochondria have been

assimilated within eukaryotic cells much later than what initially thought [10].

These revolutionary data together with the hypothesis by our and others groups about the possible existence of “extra-mitochondrial” structures in Eukaryotes with OXPHOS and ATP synthesis perfectly efficient [11-13] impose a change of paradigm in this field of studies. It is not probable that the ancestral protobacteria was engulfed just to furnish the ATP to the ancestral Eukaryote. More probably the Eukaryote, which already developed a large amount of internal membranes, was appealing for the protobacterial OXPHOS machinery. This way the OXPHOS could overpass the restraints on the transfer of ADP from cytosol to the “newborn” mitochondrion and of ATP to proto-mitochondrion to the cytosol (4 membrane passages). Indeed the presence of ATP synthase on the internal cellular membranes, reminiscent of the actual Endoplasmic Reticulum (ER), could grant the needed ATP where necessary, also to the mitochondrion, in a true symbiotic relationship. In fact, according to the classical theory [3], the Eukaryote would play a parasite role acquiring ATP from mitochondria without giving any benefits in turn. To confirm this hypothesis, the measurement of ATP concentrations in cytosol and in mitochondria that has been carried out by different groups, demonstrated that mitochondria possess less than half of ATP concentration with respect to the cytosol. This concentration gradient would allow the Adenine Nucleotide Transferase (ANT) to let ATP enter from cytosol to mitochondria and not vice versa.

### References

1. Mereschkowsky K (1910) Theory of two types of plasma as the basis of Symbiogenesis, a new theory of the genesis of the Ent - organisms. Biol Cent 30: 353-367.
2. Wallin IE (1923) The mitochondria problem. Am Nat 57: 255-261.
3. Margulis L, Bermudes D (1985) Symbiosis as a mechanism of evolution: status of cell symbiosis theory. Symbiosis 1: 101-124.
4. Lane N, Martin W (2010) The energetics of genome complexity. Nature 467: 929-934.
5. Lynch M, Marinov GK (2015) The bioenergetic costs of a gene. Proc Natl Acad Sci 112: 15690-15695.
6. Lynch M, Marinov GK (2016) Reply to Lane and Martin: Mitochondria do not boost the bioenergetic capacity of eukaryotic cells. Proc Natl Acad Sci 113: E667-E668.
7. Lane N, Martin WF (2016) Mitochondria, complexity, and evolutionary deficit spending. Proc Natl Acad Sci 113: E666.
8. Spang A, Saw JH, Jørgensen SL, Zaremba-Niedzwiedzka K, Martijn J, et al. (2015) Complex archaea that bridge the gap between prokaryotes and eukaryotes. Nature 521: 173-179.

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9. Karnkowska A, Vacek V, Zubáčová Z, Treitli SC, Petrželková R, et al. (2016) A eukaryote without a mitochondrial organelle. *Curr Biol* 26: 1274–1284.
  10. Pittis AA, Gabaldón T (2016) Late acquisition of mitochondria by a host with chimaeric prokaryotic ancestry. *Nature* 531: 101–104.
  11. Morelli A, Ravera S, Panfoli I (2011) Hypothesis of an energetic function for myelin. *Cell Biochem Biophys* 61: 179–187.
  12. Ravera S, Panfoli I, Calzia D, Aluigi MG, Bianchini P, et al. (2009) Evidence for aerobic ATP synthesis in isolated myelin vesicles. *Int J Biochem Cell Biol* 41: 1581–1591.
  13. Calzia D, Barabino S, Bianchini P, Garbarino G, Oneto M, et al. (2013) New findings in ATP supply in rod outer segments: insights for retinopathies. *Biol Cell* 105: 345–358.