REPLY TO LANE AND MARTIN:
Mitochondria do not boost the bioenergetic capacity of eukaryotic cells

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Understanding the evolution of cellular features requires a catalog of costs of building, maintaining, and operating cell parts. Lane and Martin (1) define the cost of a gene as the ratio of a cell’s metabolic rate and total gene number. This is an ecologically and evolutionarily meaningless definition, revealing nothing about the incorporation of biomass into offspring and failing to account for differences in generation lengths among organisms. We directly derive the DNA-, mRNA-, and protein-level costs of a gene, dividing these by a cell’s lifetime energy expenditure and accounting for cell division time (2). Despite Lane and Martin’s (3) claim that their paper was not about the bioenergetic costs of a gene, they stated, “By ‘energy per gene’, we mean the cost of expressing the gene” (1).

Evolutionary biology suffers from an overburden of just-so stories, and Lane (4) characterizes his description of the origin of the mitochondrion as such. However, the data are inconsistent with Lane and Martin’s claim (1) that the origin of the mitochondrion is the key to all things eukaryotic. For example, Lane and Martin stated that the mitochondrion permitted a “200,000-fold expansion in the number of genes expressed” (1). Because a typical bacterial genome contains ∼5,000 genes, this implies eukaryotic genomes harboring ∼10^9 genes, but the actual range for the latter is ∼6,000 to 40,000 (i.e., a 1.2- to 8-fold expansion).

Lane and Martin’s claims (2) that we assume “unconstrained” ATP synthesis, neglect the difference between prokaryote and eukaryote bioenergetics architecture, and neglect ribosomes are all incorrect. Our analysis used ATP consumption as a common denominator across all of cellular life, and included the cost of expressing and maintaining every cellular feature, including ribosomes. The results show that ATP requirements for growth and maintenance scale with cell volume, with complete continuity across the bacterial–eukaryote divide. Thus, the data do not support the idea that mitochondria provide an energetic boost to eukaryotic cells relative to expectations based on bacterial membranes.

The lack of increase in energetic efficiency associated with mitochondrial membranes has many potential explanations, including the fact that membranes consist of energetically expensive lipids. Many eukaryotes do not harbor numerous tiny mitochondria with maximum surface area:volume ratios, *Euglena gracilis* [incorrectly depicted in Lane and Martin (1)] being one such species. The total external surface area of mitochondria is generally <5x and often <1x the cell surface area (5–7). The surface area of internal mitochondrial membranes is generally <5x the outer-membrane area (5, 8–10), and, more importantly, only a tiny fraction (the edges of cristae) is allocated to ATP production (11). Thus, there is no evidence that membrane surface area constrains energy production.

Adopting a “eukaryocentric” view that cellular morphology, multicellularity, and debris-laden genomes are intrinsically beneficial, Lane and Martin (1) imply that the absence of a mitochondrion is the only thing preventing prokaryotic ascension to complexity. This ignores the fact that prokaryotes harbor the vast majority of Earth’s metabolic diversity, DNA, and biomass. Contrary to Lane and Martin’s claim (1) that “Mitochondria bestowed upon their host 10^7–10^9 times more power per gene,” the data suggest otherwise: In relinquishing the use of the plasma membrane for bioenergetics, eukaryotes experience no net gain in energetic capacity.


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