Debating Eukaryogenesis

Part 2: How Anachronistic Reasoning Can Lure Us into Inventing Intermediates

Dave Speijer

Eukaryotic origins are inextricably linked with the arrival of a pre-mitochondrion of alphaproteobacterial-like ancestry. However, the nature of the “host” cell and the mode of entry are subject to heavy debate. It is becoming clear that the mutual adaptation of a relatively simple, archaeal host and the endosymbiont has been the defining influence at the beginning of the eukaryotic lineage; however, many still resist such symbiogenic models. In part 1, it is posited that a symbiotic stage before uptake (“pre-symbiosis”) seems essential to allow further metabolic integration of the two partners ending in endosymbiosis. Thus, the author argued against phagocytic mechanisms (in which the bacterium is prey or parasite) as the mode of entry. Such positions are still broadly unpopular. Here it is explained why.

Evolutionary thinking, especially in the case of eukaryogenesis, is still dominated by anachronistic reasoning, in which highly derived protozoan organisms are seen as in some way representative of intermediate steps during eukaryotic evolution, hence poisoning the debate. This reasoning reflects a mind-set that ignores that Darwinian evolution is a fundamentally historic process. Numerous examples of this kind of erroneous reasoning are given, and some basic precautions against its use are formulated.

The past is a foreign country; they do things differently there.
—L. P. Hartley, The Go-Between

1. Introduction

In the first part of “Debating Eukaryogenesis,” I argued that the most likely eukaryogenic theories have to rely on positing a “pre-symbiotic” state: a state characterized by metabolic interdependency well before uptake. This constitutes an extra argument making phagocytic uptake or parasitic infection to explain endosymbiont arrival somewhat less likely. This second part of the essay tries to cast more light on why the question “How did eukaryotes come about?” is so often answered by invoking modern mechanisms (such as said processes). Phagocytosis or bacterial infection can probably be better understood as later, specialized achievements after eukaryotic ecology became available. A similar anachronistic reasoning is found in the situation where researchers hold up examples of later amitochondriate eukaryotes as models of intermediate steps toward the last eukaryotic common ancestor (LECA); see e.g. below, Section 2.1. I will try to point out the specific errors behind these kinds of reasoning, which keep on plaguing many models in evolutionary biology and constantly pop up in criticisms of non-stepwise theories that try to explain the evolution of eukaryotes.

The three most prevalent classes of anachronistic reasoning are: a) erroneously interpreting a living organism as representing a much older evolutionary intermediate, b) too easily inferring from the (prokaryotic) presence of (eukaryotic) gene homologs that they must be involved in identical processes (especially using archaeal metagenomes), and c) invoking derived, selective benefits to explain how and why ancient traits evolved. I will not return to the difficulties of inferring phenotype from genotype (class b), but refer to a paper discussing the possibility of archaeal phagocytosis as a nice illustration of the difficulties involved.[1]

Here I will try to focus on instances of such reasoning (class a/c), possibly impeding our proper understanding of eukaryogenesis. However, I will also aim at making a useful list of warning signs that we are dealing with anachronisms, and for that purpose some examples outside the main focus of eukaryogenesis will be helpful. To illustrate the lure of anachronistic reasoning I will first discuss aspects of a few publications, to be introduced in the following section. I should stress that this in no way should be interpreted as a negative view regarding the further scientific value of these publications.

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Highly-derived microbiomes in the guts of organisms represent evolutionary intermediates on the road to LECA, according to the authors (see Figure 1). Monocercomonoides species are obligate symbionts that live in the digestive tracts of insects, amphibians, reptiles, and mammals. Thus, we are asked to consider organisms living on highly-derived microbiomes in the guts of organisms representing complicated, much later stages, of eukaryotic evolution as possibly informative regarding the evolution of LECA around almost 2 billion years ago. The anachronistic error becomes even more clear if we take into account the evolutionary trajectory taken by these organisms as reconstructed by some of the same authors. First of all (and not unexpectedly, considering that LECA had a mitochondrion capable of oxidative phosphorylation; OXPHOS) these authors state: “The complete absence of mitochondria is a secondary loss, not an ancestral feature.” They are even able to give many more chronological details. Starting out with the ancestor of metamonads acquiring an anaerobic lifestyle (descendants living in anaerobic environments such as the gut), their mitochondria lost OXPHOS capabilities and only retained mitochondria-related organelles (MROs), which had lost cristae and mitochondrial genomes, and acquired further anaerobic metabolism. Under such circumstances MROs are retained because Fe-S cluster synthesis is dependent on the mitochondrial ISC pathway. The authors show that in an ancestor of Monocercomonoides, a bacterial system of sulfur utilization factors for Fe-S cluster synthesis was acquired, most likely via horizontal gene transfer (HGT). Next, the MRO could be lost completely.

Figure 1. The dangers of anachronistic reasoning. A) Asgard archaeon (brown); B) “Alphaproteobacterium-like organism” (blue); C) Cyanobacterium (green); assorted other prokaryotes are indicated in black; D) Eukaryotic multicellularity (e.g., animals); E) Archaeplastida (plants and algae). (D) and (E) come from LECA, which is schematically depicted with “bacterial” membranes, nuclei possibly formed in response to endosymbiont entry (red), and several new compartments. Some of the other later developments derived from LECA can most easily be understood as reductions (e.g., loss of oxygen as final electron acceptor leading to hydrogenosomes or complete loss of all mitochondrial structure; indicated with ???; for further details see main text). These are sometimes anachronistically interpreted as resembling organismal steps leading to LECA (??*).

2. Examples of Anachronistic Reasoning on Eukaryogenesis

2.1. A mitochondriate eukaryote capable of phagocytosis do not prove anything

Recently, an article entitled “Was the Mitochondrion Necessary to Start Eukaryogenesis?” appeared in Trends in Microbiology.[3] The authors (Hempl et al.)—who study an example of a truly completely amitochondriate eukaryote capable of phagocytosis, the oxymonad Monocercomonoides exilis—correctly highlight at the beginning of their article that this oxymonad’s existence demonstrates that “the mitochondrion is not essential for the process of phagocytosis.” At the end of their article they state under “outstanding questions,” “Truly amitochondriate eukaryotes (oxy-
momonads) should be studied in detail, namely their cell biology and energetics, as they represent a model of a putative intermediate stage of eukaryogenesis.” Something quite extraordinary has occurred: M. exilis can be seen as a model for a possible intermediate on the road to LECA, according to the authors (see Figure 1). Monocercomonoides species are obligate symbionts that live in the digestive tracts of insects, amphibians, reptiles, and mammals. Thus, we are asked to consider organisms living on highly-derived microbiomes in the guts of organisms representing complicated, much later stages, of eukaryotic evolution as possibly informative regarding the evolution of LECA around almost 2 billion years ago. The anachronistic error becomes even more clear if we take into account the evolutionary trajectory taken by these organisms as reconstructed by some of the same authors. First of all (and not unexpectedly, considering that LECA had a mitochondrion capable of oxidative phosphorylation; OXPHOS) these authors state: “The complete absence of mitochondria is a secondary loss, not an ancestral feature.” They are even able to give many more chronological details. Starting out with the ancestor of metamonads acquiring an anaerobic lifestyle (descendants living in anaerobic environments such as the gut), their mitochondria lost OXPHOS capabilities and only retained mitochondria-related organelles (MROs), which had lost cristae and mitochondrial genomes, and acquired further anaerobic metabolism. Under such circumstances MROs are retained because Fe-S cluster synthesis is dependent on the mitochondrial ISC pathway. The authors show that in an ancestor of Monocercomonoides, a bacterial system of sulfur utilization factors for Fe-S cluster synthesis was acquired, most likely via horizontal gene transfer (HGT). Next, the MRO could be lost completely.

2.2. Reduced Mitochondria or Anaerobic Lifestyles Do Not Automatically Limit Complexity

Much is made of the fact that full-scale eukaryotic complexity is retained under such circumstances; see also ref.[4]. However, it is not in the least surprising. To give just one example: blood-stream borne trypanosomes (the causative agents of sleeping sickness) divide rapidly to enormous numbers, consuming glucose without OXPHOS. These are complicated eukaryotic cells, capable of alternating variant surface glycoproteins (of which they encode a large repertoire), containing glycosomes (derived from ancestral peroxisomes) for “turbo glycolysis” and extensive, highly elaborate RNA editing in their mitochondria. I explicitly give some examples of their creativity at the molecular level, as Hempl et al. have many examples of such idiosyncrasies in strictly anaerobic eukaryotes, showing that, according to them, the development of elaborate structures does not depend on efficient mitochondrial ATP synthesis. I think my example shows that it is even the opposite: in highly derived eukaryotic environments elaborate structures evolve more easily because efficiency is not selected for (anymore). Who needs a mitochondrion in a sea of glucose? However, eukaryogenesis took place in a prokaryotic world, in which, generally, efficiency is at a premium. Misled by their highly derived anaerobic eukaryotes, living in equally highly derived environments, the authors state: “Maintaining and replicating cells is costly, but the evolution of structures does not require extra energy; therefore, it is not obvious why the mitochondrion must have preceded phagocytosis.” However, maintaining and replicating a more complex cell is more costly, so one might argue that the arrival of mitochondrial OXPHOS gave the ancestor of LECA both the energy (ATP) and the impetus (amongst others, adaptations to internal ROS formation see ref.[5]) for greater complexity, including phagocytic capabilities. In conclusion, there is indeed a difference between evolving and maintaining complex structures, but not along the lines that these authors propose. Interestingly, some of the metamonads have been seen as representatives of early amitochondriate eukaryotes before molecular analysis definitively showed they exhibit derived states, having lost their mitochondria. They thus make an unjustified comeback, with the only difference that they are not seen as real “intermediates” (which in itself discounts their own continuous evolution) anymore, but now seen as representing enlightening...
Box 1
The Contemporary Ancestor Fallacy (CAF)

Let us imagine that the first zoologists only recognized herbivores and carnivores, and were debating which type of mammal evolved first. Clearly, as herbivores could survive upon the consumption of plant material, while carnivores depended on herbivores as a food source, most zoologists opted for herbivores. To be sure about this inference our imaginary zoologists decided to open up a ruminant and a tiger. The discovery that the tiger had a much simpler gastro-intestinal tract led some of them to claim that this “simpler” system reflected the ancient state, and even that herbivores descended from carnivores. This may seem a rather glib way to discount the arguments made in the context of eukaryogenesis, but in essence it nicely illustrates the chronological flaws in this line of reasoning. Authors consistently downplay the importance of physiological/ecological considerations. How does the organism make a living? Is this a highly specialized “derived” environment? In one major respect all the single-cell eukaryotes brought up as “representatives of intermediate states” are derived, because LECA was a complex cell with a mitochondrion capable of using oxygen as the final electron acceptor. This is true for so-called “Archezoans,”[45] which were once thought to represent ancestral absence of mitochondria and peroxisomes, as well as for the Monocercomonoides species discussed in this article. The examples given are thus “reduced” eukaryotes adapted to specific niches that only became available after extensive eukaryotic diversification. Neglecting ongoing co-evolution as well as increasing ecological diversification and complexity is a hallmark of CAF.

examples of physiological possibilities. See also Box 1. Of course, the fact that in all these instances the organisms are not real representatives of intermediate stages as such does not preclude such stages occurring during eukaryogenesis. However, part 1 contains my arguments for thinking this less likely.

I mentioned the impetus of internal ROS formation, which allows me to highlight another, more hidden, anachronistic error: the tendency to assume a slow progress toward an ever more eukaryotic-like cell, which, with the uptake and slow integration of the endosymbiont, became something like LECA. In their excellent review “The Origin and Diversification of Mitochondria,”[6] Roger et al. conclude: “The endosymbiotic origin of mitochondria was of major importance to eukaryotic evolution, but it was not a single saltational event, as it is sometimes portrayed.” Of course, the alpha-proteobacterium-to-organelle transition involved thousands of evolutionary steps, so a “single event” is nonsensical. However, by focusing on direct selective pressures the merged cells might have encountered, it could be argued to have been more saltational than these authors suspect (see e.g. below, Section 2.3).

2.3. Teleological Reasoning: Invoking Current, Secondary, Selective Benefits to Explain Ancient Eukaryotic Traits

Neglecting direct selective pressures represents one of the most pernicious aspects of the anachronistic errors when debating eukaryogenesis, and one that is seldom highlighted. When explaining why many of the complex systems were positively selected, researchers tend to stress current beneficial aspects (e.g., meiotic sex allows rapid shuffling of new combinations, migration of organelar genes allows much better, fine-tuned expression, diversification of compartments in the eukaryotic cytoplasm with, e.g., lysosomes, peroxisomes, and ER allows better control over different metabolic pathways by separation). But this invites two problems. First, some of these adaptations did not have these advantages before reaching a certain level of complexity (and thus need other more immediate reasons for positive selection). Second, why did prokaryotes “wait” for about 2 billion years? One could consider the peroxisome a tell-tale organelle where eukaryogenesis is concerned. As I explained elsewhere, its evolution is nicely understood in reaction to entry of the bacterium which ended up as a mitochondrion.[7-9] Many of the hallmarks of eukaryotes can also be understood by taking into account increased endogenous ROS formation (the price to pay for ATP), and this gives us answers to both conundrums. A strong oxidative agent only became readily available to the cell after the great oxygenation event (GOE), explaining the wait. And the cell came under selective pressure to “tame” a new, internal ROS source, (partly?) explaining the increase in complexity. Part of this taming involved ever-more-efficient iron sequestration, as ROS and iron can combine to induce iron dependent oxidative damage.[10] The fact that ROS constituted both a strong selective pressure and a mutating agent might indeed have made eukaryogenesis more saltational than Roger et al. would allow. Let me point out the latest finding: the first cultivated Asgard archaeon (closest relatives to the “hosts” involved in eukaryogenesis) lives in symbioses, has metabolic pathways that could be enhanced by transferring metabolites to bacteria capable of using molecular oxygen as the final electron acceptor, and last but not least, has membrane convolutions extending into the environment, most likely optimizing metabolite exchange.[14] This last aspect also seems to make non-phagocytic uptake even more likely, and the fact that these archaea live in multispecies communities might help explain the many genes without easily identifiable archaeal or alphaproteobacterial provenance (they are referred to as being of “eukaryotic origin”).[15] This cultivated archaeon might shine a light on the origins of the highly complex, diverse, eukaryotic endomembrane system. As mentioned, it has extensions, possibly involved in metabolite exchange. They can be seen as the first stage in an inside-out model for the origins of the endomembrane
system.[16] Whether this model is correct or whether endomembrane structures arose out of bacterial outer membrane vesicles (OMVs) released by the mitochondrial ancestor into the cytosol,[17] or a combination of both is still unclear. The endosymbiotic OMVs could have diluted out the original archaeal membranes (thus explaining why eukaryotes ended up with bacterial membranes).

3. Tell-Tale Signs of Anachronistic Reasoning

3.1. Neglecting Ecological Complexity and Historical Developments

How can we easily detect anachronistic reasoning? Neglecting ongoing co-evolution as well as enhanced ecological diversification and complexity is a hallmark of anachronistic reasoning. Authors only look at the presence or absence of specific characteristics, while not giving sufficient attention to physiological/ecological considerations. There is a tendency to doubt or overlook the importance of more recent HGT, because it could explain the characteristics in question as derived.

Questions that should be asked are: “How did the organism come by its current metabolism?”. “Is this organism part of the ecology of a highly specialized ‘derived’ modern environment?”. “Can we easily come up with a scenario in which the absence or presence of a rare eukaryotic trait is a recent development instead of a 2 billion year old characteristic?”. Let us use these criteria to look at a different claim in the field of eukaryogenesis, now supporting a symbiogenic theory: the hydrogen hypothesis.[18] I have debated this theory and my alternative symbiogenic theory based on exchange of intermediate carbohydrate metabolites,[19] instead of hydrogen,[18] with William Martin before.[20,21] The argument here is whether the instances of hydrogen-producing eukaryotes are real examples of cells that have retained the capacity to synthesize hydrogen over about 2 billion years. Or could that interpretation be an example of anachronistic reasoning, when instead “normal” aerobic eukaryotes picked up the required genes later on, in hypoxic/anoxic environments (for instance while making a living as parasites)? Recent analyses come up with rather convincing reconstructions of later acquisitions in histories not unlike the one sketched for Monocercomonoides above.[22,23]

3.2. Neglecting Close Relatives and Reconstructed Ancestors

If we look at anaerobic eukaryotes we find isolated cases in quite a few deeply diverging eukaryotic clades characterized by anaerobic environments. “Hydrogenosomes” (mitochondria-related organelles capable of producing hydrogen) are also found in different phylogenetically unrelated anaerobic eukaryotes: anaerobic flagellates, chytridiomycete fungi, and several anaerobic ciliates. Many of them are found in recent, highly-diversified ecosystems. Some examples: Nycottherus ovalis, an anaerobic ciliate, lives in the hindgut of cockroaches. Blastocystis sp. (stemonopile) live in the intestinal tracts of animals. Trichomonas vaginalis is a parasite that can, for example, be found in human urogenital tracts and oral cavities. Species of the diplomonad genus Spirotrichna live under anaerobic conditions in animal intestinal tracts. Representative of a class of anaerobic fungi, the Chytridiomycetes, are found in herbivores (again, in the digestive tracts). Even considering that HGT involving eukaryotes is much more rare than prokaryotic HGT, in many cases the reconstruction of how the required HGT from bacteria present in the anaerobic environment occurred seems convincing.[21] In conclusion, the examples of anaerobic eukaryotic metabolism all seem based on several recent gene acquisitions, though this is still heavily debated.[24,25]

Importantly, the organisms in question, all seem to be derived from aerobic ancestors, having more or less normal OXPHOS, which was lost. Thus, if one proposes that anaerobic, hydrogen-producing, and aerobic metabolism coexisted in the original endosymbiotic/mitochondrion, it seems a fragile, evolutionarily unstable state: in time (let alone over the extended period under discussion here) it will cease to coexist. After a prolonged period of evolution in the presence of oxygen (and many features of LECA only make sense in the light of its presence[5,7,26]), any (possibly oxygen-sensitive) enzymes coding for anaerobic metabolic pathways must have been lost, only to reappear aided by HGT in highly-derived ecological niches. Martin and co-workers make some very interesting points regarding aerobic (mitochondrial) metabolism.[27] First of all, they stress the fact that the much-lauded efficiency gained using O2 as the final acceptor comes at a steep price, because O2 is such a strong electron acceptor. Recalculating energy efficiency in that respect shows alternative acceptors in a more favorable light. To maintain redox balances in “normal” eukaryotes uses up large amounts of energy. I would argue that this is reflected in the many “expensive” eukaryotic inventions to deal with internal ROS formation and in the fact that eukaryotic cells often opt for fermentation (which can be seen as a kind of anaerobic short-cut). Also for prokaryotes the abundant O2 coming from cyanobacteria, reflected in the GOE, proved an important selective force. The pre-GOE naphthoquinone (NQ) gave rise to the higher-redox-potential ubiquinone (Q; our mitochondrial one[8]) in aerobic bacteria. Bacteria, such as Escherichia coli, that can live aerobically and anaerobically produce both types. In a recent, beautiful paper Anand and co-workers report on the creation of E.coli that has to make do with NQ only.[28] This knock-out has a reduced growth rate, less O2 uptake and secretes lactate because of higher rates of fermentation. However, upon further adaptive evolution in the laboratory, growth rate, oxygen uptake, and lactate secretion return to wild type patterns; however, not completely. The newly evolved lines still secrete some lactate, while exhibiting slightly reduced O2 uptake and growth rates. The authors can show that reduced growth is related to higher investments in anti-oxidant measures such as the periplasmic superoxide dismutase. Thus, NQ is indeed less efficient than Q in the presence of O2, allowing more electron dissipation in the form of ROS.

Whatever the precise details, the arrival of abundant O2 and mitochondria correlated with an increase in available energy, complexity and size of eukaryotes,[5,19,29] Martin and co-workers also highlight the surprisingly low Km (in the low micromolar range, reflecting a very high affinity) of Complex IV, cytochrome c oxidase for O2, and infer that it evolved under relatively low ambient O2 concentrations.[27] This would fit with an endosymbiotic ETC able to generate ATP by OXPHOS even under low O2.
concentrations, while at the same time protecting more elaborate anaerobic host systems (possibly still present at that epoch) from oxidative damage. However, aspects of anachronistic thinking can be found in the reasons often given for dismissing the crucial role of aerobic respiration in eukaryogenesis (see ref. [30]). Here the reasoning goes: many complex eukaryotes have mitochondria but do not use O2, while many prokaryotes use oxygen but remain small and morphologically simple. I have dealt with the first part of this argument above (i.e., they constitute derived states); the second aspect can be explained by the fact that, though highly-electronegative oxygen is a perfect acceptor to generate higher amounts of ATP burning carbohydrates, amino acids, or fatty acids, this in itself is not enough. Large scale internal membrane formations (cristae) and a complex integrated metabolic network allowing the regulated oxidation of all three classes of molecules mentioned, also seem to be needed. Maybe the oxygen-using prokaryote had to end up in another cell to unlock its full potential.

4. How Did Mito-Nuclear Coadaptation, Internal ROS Formation, and Meiotic Sex Interact During Eukaryogenesis?

In Section 2.3 I mentioned the anachronistic tendency of using later, derived, selective benefits to explain the evolution of ancient eukaryotic traits. This is especially rampant in the case of meiotic sex. However, it could “simply” have evolved in the context of adaptations to oxygen, more specifically, internal ROS formation. To make the case that ROS was one of the main factors involved, a very brief introduction to mito-nuclear coadaptation, internal ROS formation, and meiotic sex is needed.

As mentioned, eukaryotic evolution seems deeply intertwined with the arrival of a pre-mitochondriion of alpha-proteobacterial ancestry and the mutual adaptation of archaeeal host and endosymbiont. One aspect of this is reflected in the term mito-nuclear coevolution (or adaptation), which refers to the fact that only a tiny, but highly important, part of the complexes involved in oxidative phosphorylation is still encoded in the organelle itself, while the large majority is encoded in the nucleus. As a result, the highly dynamic, complicated, process of manufacturing the hydrophobic inner membrane complexes involved is dependent on the compatibility of gene products of two separate genomes. These genomes differ enormously, for instance in the selective forces to which they are subjected. In theory, this might easily give rise to suboptimal or even incompatible combinations. Selection on mito-nuclear compatibility thus has been invoked as a contributor to eukaryotic evolution in general, and specifically as one of the forces behind the origin of eukaryotic (meiotic) sex.[31,32] One proposal claims that sexual recombination allows for nuclear coadaptation in the face of rapid mitochondrial mutation accumulation.[31] Indications for mito-nuclear coevolution have indeed been found,[33,34] but this does not mean it could have been a major “driving force” at the origin of meiotic sex itself or be an important rationale for its maintenance.

Other scientists (and I) think that, for example, gene migration to the nucleus as well as full meiotic sex, developing from archaeal homologous repair, can best be understood in the context of the internal, mutational, ROS formation resulting from the merger of two cells.[26,35–37] Isolated instances of gene migration would allow a gene to escape the pre-mitochondrial DNA linked to membranes full of ROS generating respiratory chain complexes, and thus be selected. During eukaryogenesis more and more genes would “move” to a compartment protected by a nuclear membrane, where they would be replicated by DNA polymerases of increasing fidelity. Such adaptations make sense in the light of endogenous ROS formation. The nucleus also allowed better (archaeeal) transcriptional control, improving over time. With systems of efficient organelle protein targeting coming into place, later organelle forming acquisitions by the descendants of LECA would exhibit similar gene trajectories (even in host cells with much better ROS control and/or organelles with lower ROS production). Crucially, the two basic values important for Muller’s ratchet: total information content (two genomes) and mutation rates (internal ROS formation), both increased in eukaryotes. This might have been enough to set the stage for meiotic sex: creating selective pressures which allowed archaeeal DNA repair mechanisms to evolve as such. An alternative hypothesis to understand aspects of eukaryotic sex also relies on the presence of mitochondria (but in this case as providers of sufficient ATP): in syncytial, (i.e., multinucleated) ancestors, different nuclei could have complemented each other, generating new chromosome combinations.[39]

5. Examples of Anachronistic Reasoning on Meiotic Sex

Mito-nuclear mate choice has been claimed to be an important component of sexual selection.[32,40] Of note, we are talking about “sexual selection” (mostly involving male–male competition and/or female choice) in the strict Darwinian sense here.[41] In this context, another anachronism rears its ugly head. Early on, almost universal uniparental mitochondrial genome inheritance was established in single-celled eukaryotes, which has unexpected consequences for the later process of sexual selection in animals (see below). These consequences are obscured because the concept of mito-nuclear compatibility is used in an overly broad, ill-defined manner, easily leading to erroneous claims here, as I indicated before, see ref. [37]. Mito-nuclear compatibility should really only refer to the compatibility of components encoded in the maternally-transmitted mitochondrion (whether RNA or protein) with nuclear-encoded components necessary for mitochondrial complexes (functional ribosomes and parts of the electron transport chain). Implicitly, this is accepted, because “hybrid” are predicted to have possible problems in all respiratory chain complexes, except Complex II (which is of course completely (!) encoded in the nucleus).[40]

Because of the extensive recombination of genetic material inherent in sexual reproduction one might wonder whether sexual selection (i.e., predicting the fitness of new combinations based on partner phenotype only) could ever work—every new combination being one of a kind. However, sexual selection, using either male–male competition or female choice, was experimentally proven in an elegant study using the flour beetle Tribolium castaneum.[42] The study indicated that mutational meltdown is prevented by meiotic reshuffling complemented by weeding out
mutations (“bad genes”) using sexual selection. The easiest way to understand this is by a direct effect of the affected gene(s) on phenotype. Adverse interactions dependent on specific combinations will of course be much harder to weed out in this fashion. This especially holds true for mito-nuclear interactions. If we assume them to be important for phenotype, and to vary sufficiently in the population, we have to conclude that they would limit sexual selection, due to uniparental inheritance. A sexually selected “healthy” phenotype might have adverse interactions with the mitochondrial genome with which it ends up, without any indication for this state of affairs being available to either males or females before partner selection. These considerations show the model in which sexual selection would allow mitonuclear compatible mate choice[32,40] to contain examples of anachronistic reasoning.

However, much more severe anachronistic, teleological, errors can be found in a recent publication that posits that sexual reproduction also evolved to prevent invasion by transmissible cancer cells.[43] As meiotic sex was fully present in single-celled LECA[36] this is a highly inappropriate way of envisaging things. The authors notice that sexual reproduction permits constant change of a multicellular organism’s genotype, which would aid detection of transmissible cancer cells by an immune system. However, this hypothesis relies on many later developments, such as multicellularity and a differentiated immune system. Thus, their proposal does not make sense. Even seeing it as an example of “later” benefit (such as concentrating on the aspect of faster probing of evolutionary space by meiotic recombination in an overarching theory of efficiently weeding out deleterious mutations[44]) leaves room for criticism. When do many organisms of necessity make the most intimate contact with members of their own species so that transmissible cancer cells can be effectively passed on? During sexual intercourse, thus demonstrating a strange circularity in reasoning.

6. Conclusions

When it comes to eukaryogenesis and eukaryotic hallmarks, proposals to reconstruct their origins from prokaryotic beginnings, using symbiogenic models, are often criticized using anachronistic examples or reasoning. I have tried to lay bare the hidden assumptions in such critiques, and to show how easily one can fall prey to neglecting the specific history of evolutionary trajectories. I hope that anachronistic reasoning will not plague the field as much in the future.

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Conflict of Interest

The author declares no conflict of interest.

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